Exhibit 15

Marc E. Wolin, Esq.
mwolin@saiber.com
John M. August, Esq.
jaugust@saiber.com
SAIBER LLC
18 Columbia Turnpike, Suite 200
Florham Park, NJ 07932
Tel: (973) 622-8401

-and-

Joseph D. Satterley, Esq.
jsatterley@kazanlaw.com
Denyse F. Clancy, Esq.
dclancy@kazanlaw.com
KAZAN, McCLAIN, SATTERLEY & GREENWOOD
A Professional Law Corporation
Jack London Market
55 Harrison Street, Suite 400
Oakland, CA 94607
Tel: (510) 302-1000

Counsel for Claimant Anthony Hernandez Valadez

IN THE UNITED STATES BANKRUPTCY COURT
FOR THE DISTRICT OF NEW JERSEY

In re:	:	Chapter 11
LTL MANAGEMENT LLC,	:	Case No. 21-30589
Debtor.	:	

DECLARATION OF RONALD F. DODSON, Ph.D., F.C.C.P., F.A.H.A.

Pursuant to 28 U.S.C. § 1746, I, Ronald F. Dodson, Ph.D., F.C.C.P., F.A.H.A., declare under penalty of perjury as follows:

1. I have personal knowledge of the facts set forth in this Declaration, except for such facts that have been made known to me in forming an opinion, in which case each such fact is of a type on which professionals in my field reasonably rely in forming such opinions. The facts stated in this Declaration that are within my personal knowledge are true. If asked, I could and

EXHIBIT 15

would testify competently to the truth and foundation for each fact and opinion asserted within this Declaration.

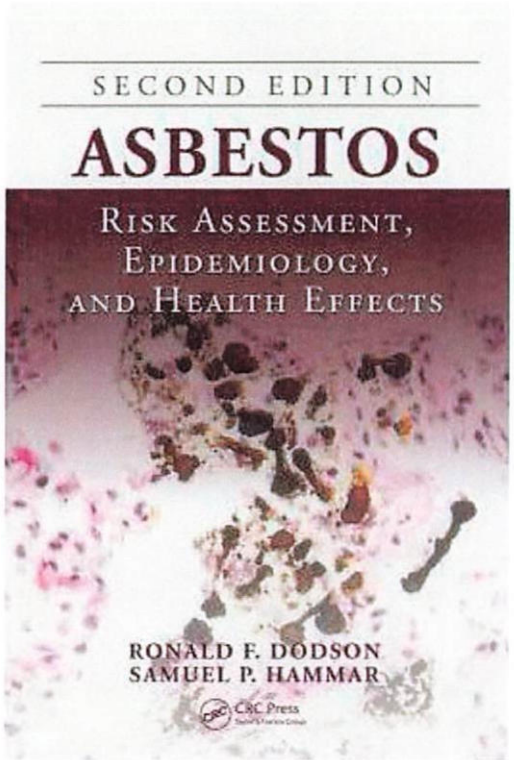
2. Attached hereto as **Exhibit 1** is a true and correct copy of my curriculum vitae, which truthfully states my qualifications to provide expert testimony in this action.

3. I received my B.A. in biology and general sciences from East Texas State College and my M.A. in biology and chemistry from East Texas State University. I received my Ph.D. from Texas A&M University, with an emphasis on biological electron microscopy. I conducted postdoctoral studies in the Department of Anatomy at the University of Texas Health Center in San Antonio. I was then appointed to the faculty of Baylor College of Medicine. After serving on the faculty at Baylor for seven years, I was recruited to the University of Texas Health Center in Tyler to begin a formal research program.

4. At the University of Texas Health Center at Tyler, I held several positions and titles, including Chief of the Department of Cell Biology and Experimental Pathology, Chairman of the Department of Cell Biology and Environmental Sciences, Associate Director for Research, Director of the Occupational/Environmental Training Division, Co-Director of the Texas Institute of Occupational Safety and Health, and Vice President for Research. I also held the position of tenured Professor of Biology at the University of Texas at Tyler.

5. My primary research focuses on determining dust levels in tissue, body fluids, and environmental samples through light and electron microscopy. My laboratories have developed some of the techniques available for preparing these samples for analysis by Analytical Transmission Electron Microscopy. I have published over 100 articles on dust-related issues and have given numerous presentations on the same topic. In addition, I co-edited two editions of a

book with Dr. Samuel Hammar entitled, Asbestos: Risk Assessment, Epidemiology, and Health Effects, currently in its second edition:



6. I retired from academia in 2005. I currently serve as President of Dodson Environmental Consulting, LLC, and as a Senior Consultant for ERI Consulting, Inc. ("ERI"). ERI provides environmental consulting services in various disciplines, including airborne contaminants. I am experienced in performing tissue digestion studies and analyzing results therefrom.

7. I am experienced in performing tissue digestion studies and analyzing results therefrom. I utilize an Analytical Transmission Electron Microscope ("ATEM") to generate tissue burden data in my studies and past publications. Some other individuals conduct tissue digestion analysis using scanning electron microscopy at low power (less than 2,000 X), whereas I prefer the utilization of ATEM at a much higher magnification which permits the detection of thinner fibers. The Asbestos Hazard Emergency Response Act designated this instrument as the "state of

the art" instrument for detection and identifying asbestos fibers. Further evaluation of the strengths in using certain instruments and the deficiency of others (including magnifications required to accurately determine the presence of asbestos fibers) is discussed in detail in the Health Effects Institute's Asbestos in Public Buildings document. [Upton, et al., *Asbestos in Public and Commercial Buildings: A Literature Review and Synthesis of Current Knowledge* (1991).] The laboratory I use for ATEM analysis is separate from my laboratory and is accredited by the National Voluntary Laboratory Accreditation Program (Federal Program) for identifying asbestos. The laboratory also is licensed by the Texas Department of State Health Services (formerly Texas Department of Health) and accredited by the American Industrial Hygiene Association.

8. In addition to my above-referenced book on asbestos, I have extensively published in peer-reviewed journals on asbestos and asbestos-related diseases—I have authored approximately 140 peer-reviewed publications and about 100 of those concerning asbestos. Many of my publications address asbestos-caused mesothelioma. I also have taught medical students about asbestos and its ability to cause disease.

9. I have reviewed the scientific literature on asbestos, elongated mineral particles, and talc fibers and their ability to cause disease. I also have published on the ability of other elongated mineral particles to cause disease.

10. I have reviewed testing of Vermont talc from many different sources, which show the presence of asbestos in that talc. Those include, but are not limited to, testing by McCrone laboratory, Cyprus laboratory, MAS, J3 Resources, Colorado School of Mines, and other internal company records confirming the presence of asbestos in talc used for Johnson's Baby Powder. For example, I have referred to the voluminous materials that I considered and relied upon in the

Hayes[1] case, which support my conclusion that Johnson's Baby Powder talc contained asbestos. Attached hereto as **Exhibits 2 and 3** are my indexes of those reliance materials from *Hayes*.

11. Additionally, as an expert consultant, I have documented the presence of asbestos and asbestiform/fibrous talc in individuals with mesothelioma, and I have opined that exposure to such structures was a cause of each individual's mesothelioma. I also have been permitted in trials to offer expert testimony on asbestos-related topics, including, among others, pathology, biology, chemistry, and microscopy. I have explained to juries that asbestos and asbestiform/fibrous talc in Johnson's Baby Powder cause mesothelioma; and how I have found asbestos, talc, and talc-associated mineral particles in the tissues of plaintiffs who developed mesothelioma due to their longtime exposures to Johnson's Baby Powder.

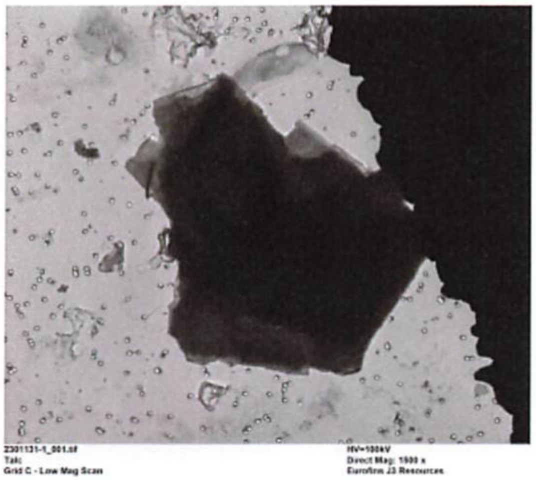
12. On May 23, 2022, I signed a declaration in this case that sets forth my causation and other opinions regarding claimant Anthony Hernandez Valadez. As more fully set forth in paragraphs 17 and 18 of my May 23rd declaration, it is my opinion, to a reasonable degree of scientific certainty, that Mr. Valadez's exposure to asbestos and asbestiform/fibrous talc in Johnson's Baby Powder is the most likely cause of his mesothelioma.

13. I have reviewed the letter dated May 14, 1971, from W.H. Ashton of Johnson & Johnson to Dr. G. Hildick-Smith regarding "Assay of Talc in BABY POWDER," a true and correct copy of which is attached hereto as **Exhibit 4**. In that letter, Mr. Ashton said that he found "talc" and "mica" minerals when he "ran an x-ray diffractograph" on a batch of Johnson's Baby Powder.

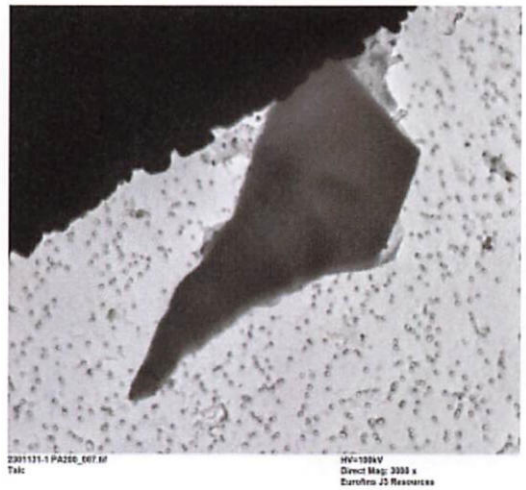
14. In addition to reviewing case-specific documents, I have also performed a particle analysis of Mr. Valadez's wet tissue to determine the composition of the non-fibrous particles in each sample. I was permitted to take half of the selected samples. The blocks chosen were defined

[1] *Hayes v. Colgate-Palmolive Co., et al.*, Jefferson, Kentucky, Circuit Court, Division Ten, Case No. 16-CI-03503.

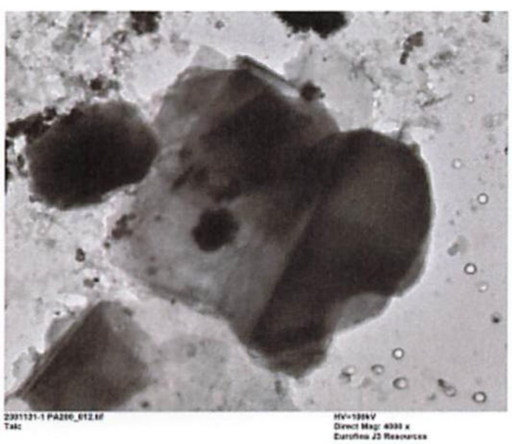
as thymus and pericardial fat, which was noted to contain some lymphatic tissue and tumor cells. [See Laboratory Surgical Pathology Report - Stanford University Medical Center, Pathology No: SHS-22-07213, Blocks A1, A3, and A4, attached hereto as **Exhibit 5**.] Talc particles and mica were found in Mr. Valadez's tissue. Below are the ATEM images of the talc particles and mica:



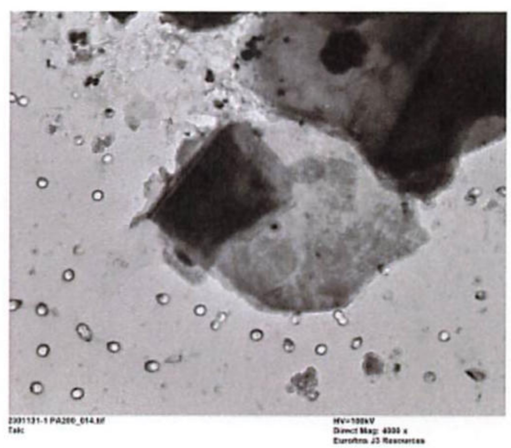
Talc



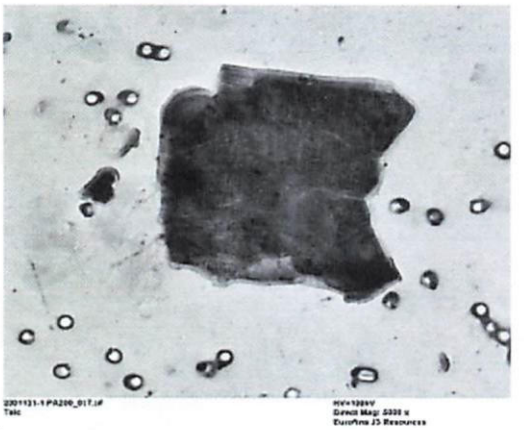
Talc



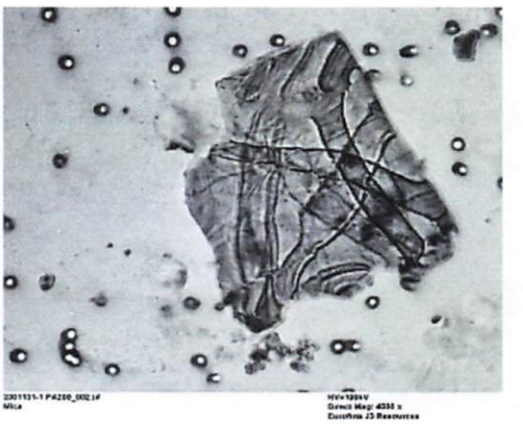
Talc



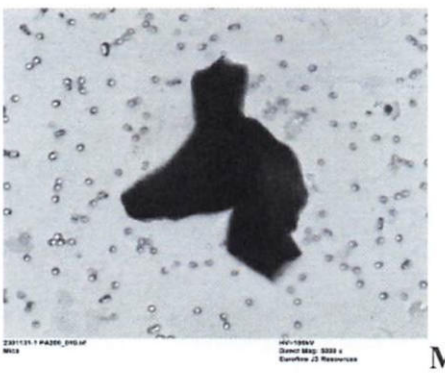
Talc



Talc



Mica



Mica

15. Based on my expertise, the scientific literature, the results of the particle analysis, and the documents I have reviewed to assess the cause of Mr. Valadez's disease, it is my opinion, to a reasonable degree of scientific certainty, that Johnson's Baby Powder was a significant contributing factor causing Mr. Valadez's mesothelioma. This is because of the following: (i) the results of the particle analysis show talc particles and mica near the tumor; (ii) Mr. Valadez's only exposures are from his and others' use of Johnson's Baby Powder talc; and (iii) Johnson's Baby Powder talc contains asbestos and asbestiform fibers.

Pursuant to 28 U.S.C. § 1746, I declare under penalty of perjury that the foregoing is true and correct to the best of my knowledge and belief. I executed this Declaration at Tyler, Texas, on February 13, 2023.

By: _____
RONALD F. DODSON, Ph.D., F.C.C.P., F.A.H.A.

KAREN DOWNING
MY COMMISSION EXPIRES
FEBRUARY 24, 2025
NOTARY ID: 12209521

Karen Downing

Exhibit 1

August 31, 2021

CURRICULUM VITAE

Ronald F. Dodson, Ph.D., F.C.C.P., F.A.H.A.

Birthdate: February 14, 1942

Birthplace: Paris, Texas

EDUCATIONAL BACKGROUND

1964 B.A., East Texas State College, Commerce, Texas

1965 M.A., East Texas State University, Commerce, Texas

1969 Ph.D., Texas A&M University, College Station, Texas

1964-65 Teaching Assistantship, East Texas State University, Commerce, Texas

1965-69 Graduate College Fellowship in Electron Microscopy, Texas A&M University, College Station, Texas

POSTGRADUATE TRAINING

1969-70 Research Associateship, Department of Anatomy, The University of Texas Health Science Center at San Antonio

ACADEMIC APPOINTMENTS

1970 Instructor, Neurology and Pathology, Baylor College of Medicine, Houston, Texas

1971-77 Assistant Professor, Department of Neurology, and Research Assistant Professor, Department of Pathology, Baylor College of Medicine, Houston, Texas

1977-78 Chief, Departments of Experimental Pathology and Environmental Sciences, The University of Texas Health Center at Tyler

1977-79 Adjunct Assistant Professor of Neurology and Pathology, Baylor College of Medicine, Houston, Texas

ACADEMIC APPOINTMENTS (Cont'd)

EXHIBIT 1

1978-86	Chief and Research Professor, Department of Cell Biology and Environmental Sciences, The University of Texas Health Center at Tyler.
1983	Acting Assistant Director for Research, The University of Texas Health Center at Tyler.
1984-85	Assistant Director for Research, The University of Texas Health Center at Tyler.
1984-2005	Professor of Biology (with Tenure), The University of Texas at Tyler (Retired August 31, 2005).
1986-99	Associate Director for Research, The University of Texas Health Center at Tyler.
1987-2004	Chairman and Professor, Department of Cell Biology and Environmental Sciences, The University of Texas Health Center at Tyler.
1989-2002	Administrative Director, Texas Institute of Occupational Safety and Health, The University of Texas Health Center at Tyler.
1989-2003	Director, Occupational and Environmental Training Program, The University of Texas Health Center at Tyler.
1999-2004	Vice President for Research, The University of Texas Health Center at Tyler.
1999-2004	Houston Endowment, Inc. Distinguished Professorship in Environmental Sciences.
1987-2005	Professor of Cell Biology and Environmental Sciences, The University of Texas Health Center at Tyler (retired-August 2005).
2005	Executive Vice President-ERI Consulting, Inc.
2006-present	Senior Consultant-ERI Consulting, Inc.
2006-present	President-Dodson Environmental Consulting, LLC.

PROFESSIONAL EXPERIENCE

1977-83	Member, Research Committee, The University of Texas Health Center at Tyler.
1979-80	Vice Chairman, Human Subjects Investigation Committee, The University of Texas Health Center at Tyler.

PROFESSIONAL EXPERIENCE (Cont'd)

1979-81	Chairman, Search Committee for Chief, Immunology/

Microbiology, The University of Texas Health Center at Tyler.

1980-81 Vice Chairman, Research Committee, The University of Texas Health Center at Tyler.

1980-83 Chairman, Human Subjects Investigation Committee, The University of Texas Health Center at Tyler.

1981-83 Secretary, Research Committee, The University of Texas Health Center at Tyler.

1982-83 Member, Committee on Animal Research Legislation, The University of Texas System.

1982-2001 Member, Animal Research Committee, The University of Texas Health Center at Tyler.

1982-1983 Member, Search Committee for Associate Director for Research, The University of Texas Health Center at Tyler.

1982-1983 Member, Search Committee for Associate Director of Clinical Affairs, The University of Texas Health Center at Tyler.

1982-1983 Chairman, Search Committee for Executive Associate Director, The University of Texas Health Center at Tyler.

1982-83 Member, The University of Texas at Tyler and The University of Texas Health Center at Tyler Health Professions Advisory Committee.

1983 Reviewer for Environmental Research

1986-92 Member, Research Advisory Committee, The University of Texas System.

1985 Reviewer for The Journal of the American Medical Association

1985 Reviewer for Archives of Pathology and Laboratory Medicine

1986-03 Member, Graduate Education Committee, The University of Texas Health Center at Tyler.

1986-89 Administrative Director, Environmental Research Institute, Tyler, Texas.

PROFESSIONAL EXPERIENCE (Cont'd)

1987 Reviewer for American Review of Respiratory Diseases

1987-91	Editorial Review Board for National Asbestos Council Journal
1988-91	Member,Board of Directors,National Asbestos Council.
1989	Director of Joint Programs for The University of Texas Health Center at Tyler and Critical Environmental Training, Inc.
1989	Adviser to Board of Directors of Critical Environmental Training, Inc.
1989	Editorial Review Board for East Texas Medicine
1990	Member, Board of Directors, Gregg Business Incubator.
.	
1991-2000	Associate Editor for Cytobios
1993-2004	Member, Internal Audit Committee, The University of Texas Health Center at Tyler.
1994-2004	Member, Residency Advisory Committee for Occupational Medicine, The University of Texas Health Center at Tyler.
1997-2003	Adjunct Member of Graduate Faculty, Stephen F. Austin State University, Nacogdoches, Texas.
1998-2001	Member, Capital Steering Committee, The University of Texas Health Center at Tyler.
1998-2002	Member, Campus Master Plan Committee, The University of Texas Health Center at Tyler.
1998-2004	Member, Compliance Program Committee, The University of Texas Health Center at Tyler.
1999-2001	Member, Board of Directors, The Discovery Science Place, Tyler, Texas.
2000-2004	Member, Defense Research Institute.
2002-2005	UTHCT-Infectious Organism Research Review Committee.
2000-Present	Member, Technology Committee of the Tyler Chamber of Commerce.
2003-2005	Member, Hazardous Materials Management Committee.
2001-2019	Member, External Advisory Board, Texas A&M University School of Rural Public Health, College Station, Texas.

PROFESSIONAL EXPERIENCE (Cont'd)

2000-2003	Peer Review Panelist, National Center for

	Environmental Research and Quality Assurance (EPA).
2003-2005	Member, Delphi Study of Asbestos Health Effects – Round I, Round II, and Round III. Co-Ordinator's of study: Daniel Banks, M.D., Jerry McLarty, Ph.D., Runhua Shi, Ph.D.
2004	International Reviewers Panel (IRP), Medical Science Monitor.
2004	Medical Science Monitor Editorial Board Reviewer.
2006	Reviewer: Journal of Toxicology and Environmental Health
2006	Member: Expert Panel on Biomarkers of Asbestos Exposure and Disease; Agency for Toxic Substances and Disease Registry: Atlanta, Georgia- May 9 and 10, 2006.
2007	Reviewer: Special Issue-Periodico di Mineralogia (Rome)
2007	Reviewer: Environmental Health Editorial
2007	External Reviewer-NIOSH Public Health Practice Project-May 2007
2007	External Peer Reviewer: EPA Draft-Final Report: "A Comparison of the Alternate Asbestos Control Method and the NESHAP Method for Demolition of Asbestos-Containing Buildings"-June 2007
2008	Peer Reviewer: CDC/NCEH/ATSDR-Project Report: "Exposure to Vermiculite from Libby, Montana at 28 Processing Sites in the United States"- March 2008
2008	Program Committee: 2008 Johnson Conference, July 14-18, 2008, Burlington, Vermont
2008	Session Chairman: Health Risks-Johnson Conference July 18, 2008, Burlington, Vermont
2008	Peer Reviewer: EPA Panel-Demonstration of Alternate Control Method Demonstration of Two Asbestos-Containing Buildings-September 11-12, 2008
2008	External Reviewer-NSF Grants
2008	Reviewer-American Journal of Industrial Medicine
2009	Reviewer-Journal of Toxicology and Environmental Health

PROFESSIONAL EXPERIENCE (Cont'd)

2009	Ad Hoc Peer Review Panel Member: Intramural

Occupational Research Agenda (NORA) FY10; The National Institute of Occupational Safety and Health (NIOSH)-May 2009

2009 Invited Participant: Workshop Sponsored by NIEHS, USEPA, ATSDR- Asbestos: A Science Based Examination of the Mode of Action Asbestos and Related Mineral Fibers-December 16-17, 2009

2010 Reviewer-Journal of Inhalation Toxicology

2011 Member: Program Committee: 2011 ASTM (D22) Johnson Conference on Asbestos

2011 Co-Chairman: Session-Human Health and Toxicology; Johnson Conference; July 25, 2011-Burlington, Vermont

LICENSURE

1993-Present Texas Department of Health Asbestos O & M Contractor

1995-Present Texas Department of Health Asbestos Operations & Maintenance Supervisor – Restricted.

1995-Present Texas Department of Health Asbestos Inspector.

1997-Present Texas Department of Health Individual Asbestos Management Planner.

PROFESSIONAL AFFILIATIONS AND RECOGNITIONS

New York Academy of Sciences.

Fellowship in the Stroke Council, American Heart Association.

Men of Achievement, 1983.

Who's Who in the South and Southwest, 1984.

Who's Who in Frontiers of Science and Technology, 1986.

Who's Who in Technology Today, 1987.

American Men and Women of Science, 1991.

Who's Who in Science and Engineering, 1993.

American Public Health Association, 1993.

PROFESSIONAL AFFILIATIONS AND RECOGNITIONS (Cont'd)

International Academy of Pathology, U.S.-Canadian Division.

Fellowship in American College of Chest Physicians.

Regional Editor - Texas Society for Electron Microscopy Newsletter 1975-77.

Editor, Southwest Science Forum Newsletter, 1976-77.

Board of Trustees - Northeast Texas Chapter, The National Multiple Sclerosis Society, 1977-81.

The National Multiple Sclerosis Society, 1977-81.

Chairman, Medical Advisory Committee, Northeast Texas Chapter.

Co-Chairman, Spring Read-A-Thon Committee, Northeast Texas Chapter, The National Multiple Sclerosis Society, 1980.

Selected as co-host for visit by National Science Foundation's exchange scientist, Dr. P. N. Viswanathan, Industrial Toxicology Research Center, Lucknow, India, March 30-April 11, 1980.

Sponsor for two-year postdoctoral study for Tomotoshi Akematsu,M.D. (Clinical Pathologist, Nishiwaki Municipal Hospital,Hyogo-Ken, Japan) 1981-83.

Sponsor for one-year postdoctoral study for Hitoshi Maeda, M.D. (Postgraduate student, Kobe University School of Medicine Kobe, Japan) 1983-84.

Selected as participant in an international workshop, co-sponsored by NATO, on the assessment of mineral content in human lungs, September 17-20, 1984, in Oxford, England.

Selected as Member, Publications Committee, National Asbestos Council, 1988-91.

Certification through U.S. Environmental Protection Agency course for "Supervision Procedures and Practices for Asbestos Abatement Projects".

Special Consultant to the Administration of The University of Texas at Austin on asbestos related problems, 1985-89.

Special Consultant to the Administration of The University of Texas System Cancer Center, Houston, Texas, on asbestos related problems, 1986-89.

Certification through U.S. Environmental Protection Agency course as asbestos inspector.

PROFESSIONAL AFFILIATIONS AND RECOGNITIONS (Cont'd)

Selected as Member of Texas Department of Health Asbestos Advisory Committee, 1987-92.

Selected as Vice-Chairman, Safety and Health Committee, National Asbestos Council, 1988-91.

Selected as Member of the Board, National Asbestos Council, Texas Chapter, 1989-96.

Selected as Member of the Advisory Committee, California Commercial Council Asbestos Committee, 1988-89.

Selected as Member of the National Asbestos Council Task Force on AHERA State Certification Reciprocity, 1988-89.

Selected as Member of Year 2000 Health Objectives for Texas, Texas Department of Health, 1989-90.

Selected to U.S. Environmental Protection Agency Atmospheric Research and Exposure Assessment Laboratory Peer Review Program, 1990.

Selected as Member of Scientific Advisory Council, Collegium Ramazzini, New York, New York, June 7-9, 1990.

Selected as Member of Steering Committee to conduct a study of higher education needs in the East and Northeast Texas region, 1993-94.

Selected as Chair, Northeast Texas Consortium of universities and community colleges, November 18, 1997.

Inducted into Hall of Honor as Distinguished Alumni in Science at Paris Junior College. Recognition was based on outstanding achievement, distinguished service, and professional leadership, November 14, 1998.

Selected as Secretary/Treasurer, Northeast Texas Consortium of universities and community colleges, September 23, 1999.

Selected as the Houston Endowment, Inc. Distinguished Professor in Environmental Sciences, August, 1999.

Selected as member, Advisory Board, Texas A&M University School of Rural Public Health, August, 2000-2016.

Selected as Executive Vice President, Board of Directors, Discovery Science Place, Tyler Texas, September, 2000.

Selected as “Eminent Scientist” & Outstanding Scholar of the Year 2001, The International Research Promotion Council.

Fellowship in American Heart Association, 2001.

PROFESSIONAL AFFILIATIONS AND RECOGNITIONS (Cont’d)

Selected as Alumni Ambassador, The 31th Annual Alumni Ambassador Forum, February 27, 2014, Texas A&M University at Commerce, College of Science, Engineering & Agriculture: Department of Biological and Environmental Sciences.

Awarded Distinguished Alumnus Paris Junior College, November 4, 2017.

Snider Lifetime Achievement Awardee 2018, Environmental Information Association, March 19, 2018 EIA National Conference, San Diego, California.

Appointed to Editorial Board, Journal of Toxicology and Environmental Health, Part B: November 2018.

GRANTS AND CONTRACTS:

Subcontract from The University of Texas Medical Branch, NIEHS Grant No. ES04147, Transplacental transfer of asbestos in humans. FY92 - $54,843.64. FY93 - $51,132.56.

Texas Department of Health Interagency Cooperative Contract No. C3000834. FY93 - $53,883.

Texas Department of Health Toxic Substance Control Division Grant #7560013546 2004, Review Asbestos Management Plans, 9/01/2003 to 8/31/2004.

Texas Department of Health Toxic Substance Control Division Grant #7560013546 2005, Review Asbestos Management Plans, 9/01/2004 to 8/31/2004.

Texas Department of Health Interagency Cooperative Contract No.75600 (3546C98-01) Floor Covering Removal. Jan. 2001

LECTURES AND PRESENTATIONS

Garcia, J.H., Dodson, R.F., Hashi, K., and Flores de Cunha, B.: Experimental cerebral infarction: Studies on permeability changes of the blood-brain-barrier. Abstract of Scientific Communication presented at the Annual Meeting, American Association of Neuropathologists, June 25-27, 1971.

Dodson, R.F. and Cheung, L.W.: Ultrastructural pathogenesis in induced cerebral infarction. Abstract of Scientific Communication presented at the Annual Meeting, Texas Academy of Science, March 16, 1973, Houston, Texas.

Dodson, R.F.: Comparative ultrastructural studies of the pathogenesis of cerebral edema in cerebral infarction and subarachnoid hemorrhage. Presented at the Cerebrovascular Clinical Research Center Workshop, Phoenix, Arizona, January

12,1973.

LECTURES AND PRESENTATIONS (Cont'd)

Dodson, R.F., Meyer, J.S., Aoyagi, M., and Hartmann, A.: Acute cerebral infarction and hypertension: An ultrastructural study. Abstract presented at the 50th Annual meeting, American Association of Neuropathologists, Boston, June 7-9, 1974.

Dodson, R.F., Chu, L.W-F, and Tagashira, Y.: Myelinated fiber response in gray matter following acute cerebral infarction, Abstract presented at the Thirty-Second Annual Meeting, Electron Microscopy Society of America, St. Louis, August 14-16, 1974.

Dodson, R.F. and Tagashira, Y.: Ependymal response in acute cerebral infarction. Abstract presented at the Thirty Second Annual Meeting, Electron Microscopy Society of America, St. Louis, August 14-16, 1974.

Dodson, R.F. and Tagashira, Y.: Ultrastructural responses of cerebral tissue following periods of ischemic insult. Abstract presented at the Fourth Annual Meeting of the Society for Neuroscience, St. Louis, October 20-23, 1974.

Tulleken, C.A.F., Meyer, J.S., Ott, E.O., Abraham, J., and Dodson, R.F.: Brain tissue pressure gradients in experimental infarction recorded by multiple wick-type transducers. Abstract presented at the Second International Symposium on Intracranial Pressure, Lund, Sweden, June 17-19, 1974.

Dodson, R.F.: Chairman, Session III, Pathology, the Thirty-Second Annual Meeting of the Electron Microscopy Society of America, St. Louis, August 14-16. 1974.

Dodson, R.F.: Chairman, Vascular Pathology Section, the Thirty-Third Annual Meeting of the Electron Microscopy Society of America, Las Vegas, August 11-15, 1975.

Dodson, R.F., Tagashira, Y., Chu, L.W-F., and Scates, R.W.: Middle cerebral artery response following occlusion with a surgical clamp. Abstract presented at the Thirty-Third Annual Meeting of the Electron Microscopy Society of America, Las Vegas, August 11-15, 1975.

Dodson, R.F., Tagashira, Y., Chu, L.W-F., and Scates, R.W.: Pericytic alterations in cerebral infarction. Abstract presented at the Thirty-Third Annual Meeting of the Electron Microscopy Society of America, Las Vegas, August 11-15, 1975.

Dodson, R.F., Tagashira, Y., Chu, L.W-F., and Scates, R.W.: Acute response of cerebral tissue following periods of ischemic insult. Abstract presented at the Thirty-Third Annual Meeting of the Electron Microscopy Society of America, Las Vegas, August 11-15, 1975.

LECTURES AND PRESENTATIONS (Cont'd)

Dodson, R.F.: Invited Lecturer - National Institutes of Health. Sponsored by the Laboratory of Neuropathology and Neuroanatomical Sciences, National Institute of Neurological and Communicative Disorders and Stroke; Igor Klatzo, M.D., Chief, Bethesda, Maryland, December 12, 1975. Seminar: Ultrastructural changes following experimental cerebral ischemia in the gerbil.

Dodson, R.F.: Invited Lecturer - Lamar University, Beaumont, Texas. Sigma Xi lecture series, March 23, 1976. Seminar: The use of animal models in the study of cerebrovascular disease in man.

Dodson, R.F., Patten, B.M. and Chu, L.W-F.: Ultrastructural change in muscle biopsy from a case of progressive ophtmalmoplegia. Abstract presented at the Thirty-Fourth Annual Meeting of the Electron Microscopy Society of America, Miami Beach, Florida, August 8-13, 1976.

Dodson, R.F., Chu, L.W-F, and Tagashira, Y.: Effects of intracarotid injections of reserpine on cerebral tissue. Abstract presented at the Thirty-Fourth Annual Meeting of the Electron Microscopy Society of America, Miami, Beach, Florida, August 8-13, 1976.

Dodson, R.F., Welch, K.M.A., and Chu, L.W-F.: Ultrastructural changes following experimental cerebral ischemia in the gerbil. Abstract presented at the Sixth Annual Meeting of the Society of Neuroscience, Toronto, Ontario, Canada, November 7-11, 1976.

Dodson, R.F., Welch, K.M.A., and Chu, L.W-F.: Cytoarchitectural changes in brains of ischemic gerbils. Abstract presented at the Cerebrovascular Clinical Research Center Workshop, Miami, Florida, February 23-24, 1977.

Dodson, R.F.: Invited Testimony - Submitted to the Commission for Control of Huntington's Disease and Its Consequences, Regional Meeting for Arkansas, Louisiana, Oklahoma and Texas, held May 3, 1977, at The University of Texas Health Science Center, Dallas, Texas, sponsored by National Institutes of Health, Bethesda, Maryland.

Dodson, R.F., Chu, L.W-F., and Ishihara, N.: Cerebral tissue response in chronic electrode implantation. Abstract presented at the Joint Meeting of Electron Microscopy Society of America and the International Conference on X-Ray Optics and Microanalysis, Boston, Massachusetts, August 22-26, 1977.

LECTURES AND PRESENTATIONS (Cont'd)

Dodson, R.F., Chu, L.W-F., and Miyakawa, Y.: Cerebral response to ischemia induced by the embolism model. Abstract presented at the Joint Meeting of Electron Microscopy Society of America and International Conference on X-Ray Optics and Microanalysis, Boston, Massachusetts, August 22-26, 1977.

Dodson, R.F.: Invited lecturer - Texas Eastern University, Tyler, Texas, School of Science and Mathematics Seminar Series, September 21, 1978. Seminar: The application of animal models to the understanding of ultrastructural pathogenesis of human diseases.

Dodson, R.F., Williams, M.G. Jr., and Hurst, G.A.: Neutrophil response to intratracheally injected asbestos. Abstract presented at the Joint Meeting of Electron Microscopy Society of America and Microbeam Analysis Society, San Antonio, Texas, August 13-17, 1979.

Williams, M.G. Jr., Dodson, R.F., and Hurst, G.A.: A Technique for Studying Single Cells by Light, Transmission and Scanning Microscopy. A presentation at the Joint Meeting of Electron Microscopy Society of America and Microbeam Analysis Society, San Antonio, Texas, August 13-17, 1979.

Dodson, R.F.: Moderator and Coordinator - Symposia on Asbestos in Schools, jointly sponsored by The University of Texas Health Center at Tyler, (Departments of Cell Biology and Environmental Sciences and Epidemiology/Biomathematics), Texas Department of Health, Region 7, and the Texas Air Control Board, Air Quality Control Region 12; held at The University of Texas Health Center at Tyler, Tyler, Texas, September 19, 1979.

Dodson, R.F.: Guest Faculty, Invited Lecturer. Electromicroscopic study of experimental asbestosis: The coated and uncoated fibers. Presented in 3-day Symposia on Asbestos Associated Diseases October 11-13, 1979, at Baylor College of Medicine, Houston, Texas, sponsored by The Office of Continuing Education, Baylor College of Medicine, Houston, and co-sponsored by The American Society of Clinical Pathologists and The Houston Society of Clinical Pathologists.

Dodson, R.F., Hurst, G.A., and Williams, M.G., Jr.: Short-Term Response of Lung Parenchyma Following "Amosite" Asbestos Exposure. Presented at the American Thoracic Society/ Canadian Thoracic Society Meeting, Environmental and Occupational Health Assembly, Washington, D.C., May 19, 1980.

LECTURES AND PRESENTATIONS (Cont'd)

O'Sullivan, M.F., Williams, M.G., Jr., and Dodson, R.F.: Lung response to asbestos. Presented at Fall Meeting of Texas Society for Electron Microscopy, College Station, Texas, October 9-11, 1980.

Piers, D.O., Williams, M.G., Jr., and Dodson, R.F.: Pulmonary free cell response to asbestos as determined from lavage preparations. Presented at Fall Meeting of Texas Society for Electron Microscopy, College Station, Texas, October 9-11,1980.

Dodson, R.F., Williams, M.G., Jr., McLarty, J.W., and Hurst, G.A.: Particulate matter in the sputum from former asbestos workers: An ultrastructural study. Clin. Research, December, 1980. Abstract.

Martin, R.R. and Dodson, R.F.: Human pulmonary alveolar macrophages phagocytize ash from Mt. St. Helens with release of chemotactic factors. Presented at the American Thoracic Society Meeting, Detroit, Michigan, May 10-18, 1981.

Dodson, R.F., Williams, M.G., McLarty, J.W. and Hurst, G.A.: An ultrastructural study of particulate matter and ferruginous bodies in the sputum from former asbestos workers. Presented at Electron Microscopy Society of America, Atlanta, Georgia, August 11, 1981.

Dodson, R.F.: Invited Lecturer -- Stephen F., Austin State University, Nacogdoches, Texas, Biology Department Graduate Seminar, February 24, 1982. Seminar: Asbestos - understanding the problem.

Dodson, R.F., and Martin, R.R.: In Vitro interactions of human pulmonary macrophages with volcanic ash. Presented at International Academy of Pathology Meeting, Boston, Massachusetts, March 1-5, 1982.

Dodson, R.F., O'Sullivan, M.F., Williams, M.G. and Hurst, G.A.: Analysis of cores of ferruginous bodies from former asbestos workers. Presented at International Conference on Occupational Lung Disease, Chicago, Illinois, March 24-27, 1982.

Dodson, R.F.: Invited Lecturer -- The University of Texas at Tyler, Tyler, Texas, May3-4, 1982. Lecture given at Pathophysiology course for nurses.

Williams, M.G., Corn, C., Dodson, R.F., and Hurst, G.A.: Isolation of asbestos from lung tissue and sputum. Presented at Electron Microscopy Society of America, Washington, D.C., August 10, 1982.

LECTURES AND PRESENTATIONS (Cont'd)

O'Sullivan, M.F., Corn, C., Dodson, R.F., and Hurst, G.A.: The influence of inflation level on the ultrastructure of pleura. Presented at Electron Microscopy Society of America, Washington, D.C., August 10, 1982.

Dodson, R.F.: Invited Lecturer -- Baylor College of Medicine, Houston, Texas, Department of Pathology Monthly Research Seminar, December 15, 1982. Seminar: Asbestos: Something Old/Something New.

Dodson, R.F., Greenberg, S.D., Williams, M.G., and Corn, C.: Ferruginous body content from lung tissue of occupationally and non-occupationally exposed groups. Presented at International Academy of Pathology Meeting, Atlanta, Georgia, February 28, 1983.

Dodson, R.F.: Invited Lecturer -- The University of Texas at Tyler, Tyler, Texas, March 8-9, 1983. Lecture given on Pathological Responses of the Lung at Pathophysiology course for nurses.

Dodson, R.F.: Invited Lecturer -- The University of Texas at Tyler, Tyler, Texas, May 2-3, 1983. Lecture given on Cancer at Pathophysiology course for nurses.

Lawrence, E.C., Fox, T.B., Hall, B.T., Putman, M., Greenberg, S.D., Mace, M.L., Dodson, R.F. and Martin, R.R.: Effects of amosite asbestos on human pulmonary alveolar macrophage functions. Presented at American Thoracic Society Meeting, Kansas City, Missouri, May 8, 1983.

Dodson, R.F., Akematsu, T., Williams, M.G., O'Sullivan, M.F., and Hurst, G.A.: Acute response of lung parenchyma to volcanic ash. Presented at Electron Microscopy Society of America, Phoenix, Arizona, August, 1983.

Ford, J.O., Dodson, R.F., Williams, M.G., and Hurst, G.A.: The blood/air barrier as defined by horseradish peroxidase in the normal guinea pig lung. Presented at Electron Microscopy Society of America, Phoenix, Arizona, August, 1983.

Davis, M.L., Lewandowski, J., Dodson, R.F., and Hurst, G.A.: Ultrastructure of the bronchiolar epithelium in the guinea pig. Presented at Electron Microscopy Society of America, Phoenix, Arizona, August, 1983.

Ford, J.O., and Dodson, R.F.: Endocytosis of amosite asbestos by Paramecium Multimicronucleatum. Presented at Texas Society for Electron Microscopy Meeting, Tyler, Texas, October, 1983.

LECTURES AND PRESENTATIONS (Cont'd)

Davis, M.L., and Dodson, R.F.: Some SEM observations of the early pulmonary response to "Amosite" asbestos exposure in the guinea pig. Presented at Texas Society for Electron Microscopy Meeting, Tyler, Texas, October, 1983.

Ford, J.O., Dodson, R.F., and Williams, M.G.: An ultrastructural study of the blood/air barrier in the guinea pig. Poster presented at Texas Society for Electron Microscopy Meeting, Tyler, Texas, October, 1983.

Dodson, R.F., Williams, M.G., O'Sullivan, M.F., Corn, C.J., Hurst, G.A.: Quantitation of uncoated asbestos fibers and ferruginous bodies in lungs of asbestos workers. Presented at International Academy of Pathology Meeting, San Francisco, California, March 12, 1984.

Hurst, G.A., McLarty, J.W., Dodson, R.F.: The significance of pleural calcification in amosite asbestos workers. Presented at Fifth European Congress of Diseases of the Chest, Lisbon Portugal, June 4-7, 1984.

Dodson, R.F., Williams, M.G., O'Sullivan, M., Corn, C.J., Hurst, G.A., Greenberg, S.D.: Quantitation of uncoated asbestos fibers and ferruginous bodies in lungs of asbestos workers. Presented at International Congress, Miami, Florida, September 6, 1984.

Dodson, R.F., Williams, M.G., Corn, C.J., Hurst, G.A.: Asbestos bodies, uncoated fibers, and other particulates in nonurban lung cancer patients. Presented at The University of Texas Graduate School of Biomedical Sciences in Houston, Texas, September 27, 1984.

Davis, M.L., Lewandowski, J., Dodson, R.F.: A morphological study of the distal airways. Presented at The University of Texas Graduate School of Biomedical Sciences in Houston, Texas, September 27, 1984.

Davis, M.L., Ford, J.O., Dodson, R.F.: Preparation of guinea pig airways for electron microscopy. Presented at the meeting of the Texas Society for Electron Microscopy, San Antonio, Texas, April 12, 1985.

Dodson, R.F., Maeda, H., Williams, M.G., Ford, J.O.: Long term pulmonary effects of diatomaceous earth in adult guinea pigs. Presented at the meeting of the American Thoracic Society, Anaheim, California, May 14, 1985.

Dodson, R.F., O'Sullivan, M.F., Corn, C.J., Williams, M.G.: Nonasbestos ferruginous bodies in man. Presented at the meeting of the Electron Microscopy Society of America, Louisville, Kentucky, August 5, 1985.

LECTURES AND PRESENTATIONS (Cont'd)

Davis, M.L., Ford, J.O., Dodson, R.F.: Ultrastructure of major conducting airway epithelium in the guinea pig. Presented at the meeting of the Electron Microscopy Society of America, Louisville, Kentucky, August 5, 1985.

Corn, C.J., Williams, M.G., Dodson, R.F.: An analysis of residual asbestos remaining in preparative vials following bleach digestion. Presented at the meeting of the Electron Microscopy Society of America, Louisville, Kentucky, August 5, 1985.

DeShazo, R.D., Diem, J.E., Banks, D., Chapman, Y., Bozelka, B., and Dodson, R.F.: Asbestos inhibits natural killer cell response to interferon. Presented at the Southern Society for Clinical Investigation Meeting, New Orleans, Louisiana, February 5, 1986.

Dodson, R.F.: Particulate analysis samples from amosite workers. Presented at the joint meeting of the Electron Microscopy Society of America/Microbeam Analysis Society Albuquerque, New Mexico, August 14, 1986.

Dodson, R.F: Invited Lecturer -- Presentation for the Asbestos Programs Group, Environmental Health and Safety Division, Georgia Tech Research Institute, Oklahoma City, Oklahoma, August 26, 1986.

Dodson, R.F.: Invited Lecturer -- Health Effects of Asbestos Presentation for the National Conference of State Legislatures' Seminar on Asbestos Safety in Buildings, Kansas City, Missouri, September 12, 1986.

Kennedy, T., Melnicoff, P., Dodson. R., Rawlings, W. Hoidal, J.: Kaolin catalyzes hydroxyl radical generation from hydrogen peroxide. Presented at the American College of Chest Physicians' Third International Conference on Environmental Lung Disease, Montreal, Quebec, Canada, October 15-18, 1986.

Garcia, J.G.N., Callahan, K., Davis, L., Johnson, A.R., Corn, C., Dodson, R.F.: Activation of Human umbilical vein endothelium following phagocytosis of asbestos and fiberglass particles. Presented at the American College of Chest Physicians' Third International Conference on Environmental Lung Disease, Montreal, Quebec, Canada, October 15-18, 1986.

Corn, C.J., Williams, M.G., Dodson, R.F.: An electron microscopic analysis of residual asbestos remaining in preparative vials following bleach digestion. Presented at the meeting of the Texas Society for Electron Microscopy, Houston, Texas, October 16-18, 1986.

LECTURES AND PRESENTATIONS (Cont'd)

Kennedy, T.P., Dodson, R., Rawlings, W. and Hoidal, J.R.: Kaolin catalyzes hydroxyl radical generation from hydrogen peroxide. Presented at the American Federation for Clinical Research, New Orleans, Louisiana, January 28-30, 1987.

Dodson, R.F., Hurst, G., Williams, M., Corn, C., and Greenberg, S.: A comparison of light and electron microscopy techniques for defining occupational exposure to asbestos. Presented at the meeting of the International Academy of Pathology, Chicago, Illinois, March 9-13, 1987.

Garcia, J.G.N., Callahan, K.S. Gray, L.D., Azghani, A., Johnson, A.R., and Dodson, R.F.: Amosite asbestos is toxic to vascular endothelium and induces alterations in endothelial cell function. Presented at the meeting of the American Thoracic Society, New Orleans, Louisiana, May 10-13, 1987.

Kennedy, T.P., Ky, H., Roa, N.V., Hopkins, C., Tolley, E., Dodson, R., and Hoidal, J.R.: Asbestos and kaolin cause red cell hemolysis by acting as fenton reagents. Presented at the meeting of the American Thoracic Society, New Orleans, Louisiana, May 10-13, 1987.

Dodson, R.F.: Keynote Speaker -- Health Effects of Asbestos presented to the National Conference of State Legislatures. Indianapolis, Indiana, July 27, 1987.

Dodson, R.F.: Invited Lecturer -- An Interdisciplinary Training Program for Supervision of Procedures and Practices of Asbestos Abatement sponsored by the Texas Engineering Extension Service of the Texas A&M University System, El Paso, Texas, August 10, 1987.

Dodson, R.F.: Invited Lecturer -- Asbestos symposium sponsored by Asbestos Programs Group, Environmental Health and Safety Division, Georgia Tech Research Institute, Atlanta, Georgia, August 17, 1987.

Dodson, R.F.: Keynote Speaker -- Asbestos Substitutes: A Growing Concern. National Asbestos Council Third Annual Fall Technical Asbestos Abatement Conference and Exposition, Oakland, California, September 22, 1987.

Dodson, R., Williams, M., Corn, C., Idell, S., and McLarty, J.: Asbestos fibers in sputum--an indicator of past occupational exposure. Presented at the meeting of the United States and Canadian Academy of Pathology, Washington, D. C., February 28-March 4, 1988. Lab.Invest. 58:144, 1988.

Garcia, J.G.N., Griffith, D.E., Garcia, P.L., Iriana, S., Dodson, R.F., and Callahan, K.S.: Alveolar macrophages from asbestos-exposed workers are primed to release LTB_4. Presented at the meeting of the American Thoracic Society,

Las Vegas, Nevada, May 11, 1988.

LECTURES AND PRESENTATIONS (Cont'd)

Dodson, R.F.: The lung as a mirror for past exposure to asbestos and other dusts. Presented at the 1988 Fall Technical Conference and Exposition of the National Asbestos Council, Inc., Boston, Massachusetts, September 20, 1988.

Ford, J.O. and Dodson, R.F.: Pulmonary response following a second asbestos exposure. Presented at the meeting of Texas Society for Electron Microscopy, Galveston, Texas, October 6-8, 1988.

Dodson, R.F.: Why are dusts toxic? Presented at the Sixth Annual Asbestos Abatement Conference and Exposition of the National Asbestos Council, Inc., Anaheim, California, March 30, 1989.

Dodson, R.F.: Invited Lecturer -- Collegium Ramazzini Workshop on "Disease Potential of Different Asbestos Fiber Varieties," Ottawa, Canada, March 20-22, 1989.

Dodson, R.F.: Invited Lecturer -- Manmade mineral fiber update. Presented at National Insulation Contractors' Association Convention. Charleston, South Carolina, April 9-12, 1989.

Griffith, D.E., Dodson, R.F., Garcia, J.G.N., Levin, J., and Kronenberg, R.S.: Airflow obstruction in asbestos and silica exposed workers. Presented at the meeting of the American Thoracic Society, Cincinnati, Ohio, May 17, 1989.

Dodson, R.F.: Application of electron microscopy in analysis. Presented at the Baylor College of Medicine seminar entitled "The Pathology of Occupational Lung Disease: An Update," Houston, Texas, May 19, 1989.

Dodson, R.F.: Invited Lecturer -- Asbestos particles (coated and uncoated) in the lungs. Presented at the Baylor College of Medicine Pathology/Radiology Conference, Houston, Texas, August 23, 1989.

Dodson, R.F.: Health effects of manmade mineral fibers - questions remain. Presented at the 1989 Fall Technical Conference and Exposition of the National Asbestos Council, Indianapolis, Indiana, September 27, 1989.

McKee, T.R., Crossman, R.N., Dodson, R.F., Jones, C., Walker, G. and Tompkins, R.: Applications of microscopy techniques and their impact on the asbestos industry. Presented at the Seventh Annual Asbestos Abatement Conference and Exposition of the National Asbestos Council, San Antonio, Texas, February 20, 1990.

Kronenberg, R., Levin, J. and Dodson, R.F.: Asbestos health issues in the nineties. Presented at the Seventh Annual Asbestos Abatement Conference and Exposition of the National Asbestos Council, San Antonio, Texas, February 20, 1990.

LECTURES AND PRESENTATIONS (Cont'd)

Dodson, R.F.: Invited Lecturer -- Comparison of fiber burdens of parenchyma, lymph nodes and pleural plaques in shipyard workers. Presented at Collegium Ramazzini, "The Third Wave of Asbestos Disease: Exposure to Asbestos in Place," New York, New York, June 7, 1990.

Levin, J., Shepherd, R., Kronenberg, R. and Dodson, R.: Making the diagnosis: asbestos and benign pleural disease. Presented at the 1990 Fall Technical Conference and Exposition of the National Asbestos Council, Phoenix, Arizona, September 12, 1990.

Fraire, A.E., Greenberg, S.D., Roggli, V.L., Cartwright, J., Dodson, R., Williams, G. and el-Naggar, A.: Light microscopic, ultrastructural and flow cytometric findings in rat pleural mesothelial cells following crocidolite asbestos inoculation. Presented at the American College of Chest Physicians 56th Annual Assembly, Toronto, Ontario, Canada, October 22-26, 1990, Chest 98:66S.

Dodson, R.F., Williams, M.G., Corn, C.J., Brollo, A. and Bianchi, C.: Nonasbestos fiber burden in individuals exposed to asbestos. Presented at the NATO Advanced Research Workshop on Mechanisms in Fibre Carcinogenesis, Albuquerque, New Mexico, October 21-26, 1990.

Friedman, W., Ewing, W., and Dodson, R.F.: Asbestos fiber length - current issues. Presented at National Asbestos Council 8th Annual Asbestos Management Conference and Exposition, New Orleans, Louisiana, February 19-22, 1991.

Dodson, R.F., Ewing, E., Ewing, W., Oppenheim-McMullen, J., and Millette, J.: Assessing risk in buildings containing asbestos. Presented at American Public Health Association 119th Annual Meeting, Atlanta, Georgia, November 13, 1991.

Dodson, R.F., O'Sullivan, M., Corn, C.J., Garcia, J.G.N., Stocks, J.M., and Griffith, D.E.: Nonasbestos ferruginous bodies in bronchoalveolar lavage. Presented at American Thoracic Society International Conference, Miami, Florida, May 18, 1992.

Fraire, A.E., Greenberg, S.D., Spjut, H.J., Roggli, V.L., Dodson, R., Cartright, J. and Williams, G.: Histopathologic characterization of rat pleural mesothelium following intrapleural inoculation with fiber glass. Presented at XIX International Congress of the International Academy of Pathology, Madrid, Spain, October 18-23, 1992.

LECTURES AND PRESENTATIONS (Cont'd)

Fraire, A.E., Greenberg, S.D., Spjut, H.J., Cartright, J. Roggli, V.L., Dodson, R., and Williams, G.: Histopathologic, ultrastructural and flow cytometric characterization of fiberglass-induced pleural mesothelioma in the fischer 344 rat. Presented at XVII World Congress on Diseases of the Chest, Amsterdam, June 13-18, 1993.

Aust, A.E., Lund, L.G., Williams, M.G., and Dodson, R.F.: Iron associated with asbestos bodies is responsible for the formation of single-strand breaks in øX174 RFI DNA. Presented at Oxygen Radicals and Lung Injury Conference, Morgantown, West Virginia, August 30 to September 2, 1993.

Fraire, A.E., Greenberg, S.D., Spjut, H.J., Roggli, V.L., Dodson, R. F, Williams, G. and Baker, S.: Erionite induced pleural changes in the Fischer 344 rat. Presented at the ALA/ATS International Conference, Boston, Massachusetts, May 22-25, 1994.

Dodson, R.F., O'Sullivan, M., and Corn, C.J.: The ratio of ferruginous body content to the types of uncoated asbestos fibers in occupational and paraoccupational cohorts. Presented at American Thoracic Society International Conference, Seattle, Washington, May 24, 1995.

Dodson, R.F. and Hammar, S.P.: Invited Lecturer -- Asbestos fiber burden in lung tissue of patients with malignant mesothelioma in the Northwest United States. Presented at the Society for Ultrastructural Pathology meeting, Washington, D.C., March 24, 1996.

Levin, J.L., Frank, A.L., Rountree, P.P., Shepherd, J.R., Dodson, R.F., and Crossman, R.: Invited Lecturer -- The role of electron microscopy. In Postgraduate Seminar: Occupational Pneumoconioses Update. Presented at the American Occupational Health Conference, San Antonio, Texas, April 26-May 3, 1996.

Frank, A.L., Dodson, R.F., and Williams, M.G.: Lack of tremolite in UICC reference chrysotile and the implications for carcinogenicity. Presented at the Inhaled Particles VIII Meeting, Cambridge, England, August 26-30, 1996.

Fraire, A.E., Dodson, R.F, Williams, G., Dickson, E. and Corn, C.: Do asbestos (ferruginous bodies form in extrapulmonary sites? An experimental study in a guinea pig model. Presented at the XXI International Congress of the International Academy of Pathology, Budapest, Hungary, October 20-25, 1996.

LECTURES AND PRESENTATIONS (Cont'd)

Levin, J., O'Sullivan,M., Corn, C., and Dodson, R.: Ferruginous bodies formed predominantly on chrysotile cores. Presented at the Third International Symposium, *Impact of Cancer Biotechnology on Diagnostic and Prognostic Indicators in Predictive Oncology and Therapy* in Nice, France, October 26-28, 1996.

Atkinson, M.A.L., Tate, R., Williams, M.G., and Dodson, R.: Erionite, a fibrous zeolite, binds iron and induces DNA single-strand nicks. Presented at 6th International Congress on Cell Biology and 36th American Society for Cell Biology Annual Meeting, San Francisco, California, December 7-11, 1996, Molecular Biology of the Cell 7:144A.

Williams, M.G., Crossman, R.N., Dodson, R.F.: Results from a search for tremolite asbestos in UICC chrysotile B. Presented at the EIA Meeting, New Orleans, Louisiana, March 25, 1997.

Dodson, R.F., O'Sullivan, M.F., Huang, J., Hammar, S.P.: Invited Lecturer -- Asbestos in extrapulmonary sites-omentum and mesentery tissue. Presented at 6th International Conference on Environmental & Occupational Lung Disease in Vancouver, British Columbia, Canada, February 11, 1999.

Hammar, S.P., Dodson, R.F.: Pulmonary Granulomatosis in Association Intraparencymal Birefringent Material. Presented at Pulmonary Pathology Meeting and Medical Symposium, Asheville, North Carolina, August 25-27, 1999.

Dodson, R.F.: The role of fiber analysis in the diagnosis and attribution of asbestos-related diseases. Presented at Pulmonary Pathology Meeting and Medical Symposium, Asheville, North Carolina, August 25-27, 1999.

Dodson, R.F.: The medical and scientific aspects in the quantitative analysis of ferruginous bodies and asbestos fibers. Present at Defense Research Institute's Asbestos Personal Injury Litigation Seminar, November 2-3, 2000

Williams, M.G., Crossman, R., Dodson, R.F.: Asbestos Release During Floor Tile Removal. Presented at EPA Conference on Asbestos Health Effects, Oakland, California – May 23-26, 2001.

Dodson, R.F., Atkinson, M.A.L: Invited Presentation: Asbestos Burden in Tissue: Little Things Mean a Lot. Presented at the ASTM Johnson Conference, Burlington, Vermont, July 18-22, 2005.

Dodson, R.F., Invited Presentation: Extrapulmonary Exposure to Asbestos, First Meeting of the Committee on Asbestos: Selected Health Effects, Institute of Medicine-Division of Population Health and Public Health Practice; Washington, DC; July 26, 2005.

LECTURES AND PRESENTATIONS (Cont'd)

Dodson, R.F., Invited Speaker: Measurements of Asbestos Fibers in Tissues: Presented in the Session: Asbestos and Man-made Mineral Fibers: Conference-Framing the Future in the Light of the Past: Living in a Chemical World 2005-Third International Scientific Conference; Collegium Ramazzini; Bologna, Italy; September 18-21, 2005.

Dodson, R.F. Invited Speaker: Asbestos Medicine-A Prognosis on the Future of Asbestos Disease: Presented-Mealey's National Asbestos Litigation Conference, Boston, Mass. September 18, 2006.

Dodson, R.F. Keynote Address: Asbestos and Human Health: Something Old, Something New, and a Lot of Unknowns: Presented at the Twenty-fifth Anniversary Meeting of the Environmental Information Association, Albuquerque, New Mexico. March 17, 2008.

Dodson, R.F. Presentation: Mesothelioma-What We Know Based on Tissue Analysis for Asbestos, Johnson Conference, Burlington Vermont, July 18, 2008.

Dodson, R.F. Invited Witness-Topic: Asbestos and Mesothelioma; State of Texas Senate Committee for State Affairs, Austin, Texas; March 23, 2009.

Dodson, R.F. Invited Panel Member and Speaker: The Physical Aspects of Asbestos/Elongated Minerals and Reaction with Tissue. Panel 1: Toxicology: Workshop on the NIOSH Research Roadmap on Asbestos Fibers and Other Elongated Mineral Particles; Institute of Medicine-National Research Council, National Academy of Sciences; Washington D.C. March 30,2009.

Dodson, R.F. Invited Speaker: Fiber Burden Analysis-The What's The Why's and The How's; National Asbestos Litigation Conference, September 23-25, San Francisco, California.

Dodson, R.F. Invited Panel Member: Exposure Team; Asbestos: A Science-Based Examination of the Mode of Action of Asbestos and Related Mineral Fibers; Sponsored by National Institute of Environmental Health Sciences, NIEHS Superfund Research Program, U.S. Environmental Protection Agency, Agency for Toxic Substances and Disease Registry. December 16-17,2009; Chapel Hill, North Carolina.

Dodson, R.F. Presentation: Observations from Quantitative Analysis of Tissue Burden for Elongated Particles by Light and/or Transmission Electron Microscopy as Relate to Attribution of Mesothelioma, ASTM-Johnson Conference, Burlington Vermont July 25, 2011

LECTURES AND PRESENTATIONS (Cont'd)

Dodson, R.F. Invited Presentation: Identification and Quantification of Asbestos Burden; in Current Concepts and Controversies in Asbestos-Related Disease; Massachusetts General Hospital/Department of Pathology Harvard Medical School, May 5-6, 2012; Boston, Massachusetts.

Dodson, R.F. Presentation: Asbestos in air and tissue samples-Now you see them. Now you don't! Texas EIA Technical Seminar; September 28, 2012; Houston, Texas

Dodson, R.F. Invited Presentation: "What Can Be Learned from Tissue Burden of Elongated Mineral Particles"! Workshop: Mineral Fibers in the Upper Midwest. EPA Mid Continent Laboratories in Duluth, MN. October 6-7, 2015

Poye, L.W. and Dodson RF. Invited Presentation: Variables in Identification of Tissue Burden of Asbestos Fibers in Human Tissue Based on Preparation, Instrumentation and Magnification: Collegium Ramazzini, Carpi, Italy October 27, 2016.

LECTURER FOR EPA AND OSHA TRAINING COURSES

Dodson, R.F.: Lecturer -- Health Effects of Asbestos Exposure. Presentations for the Texas Engineering Extension Service of The Texas A&M University System Asbestos Training Programs, 1986-1992:

1. An Interdisciplinary Training Program for Supervision of Procedures and Practices of Asbestos Abatement.

2. Asbestos Worker Training Program

3. Asbestos Inspector/Manager Training Course.

Dodson, R.F.: Lecturer -- Health Effects of Asbestos Exposure. Presentations for The University of Texas Health Center at Tyler and Critical Environmental Training, Inc. Asbestos Training Programs, 1988, 1989:

1. An Interdisciplinary Training Program for Practices and Procedures of Asbestos Abatement for Supervisors and Contractors.

2. An Interdisciplinary Training Program for Building Inspector for Asbestos Abatement Projects.

3. NIOSH 582 7400 Method - ORM (Air Sampling Course).

LECTURER FOR EPA AND OSHA TRAINING COURSES (Cont'd)

Dodson, R.F.: Lecturer -- Health Effects of Asbestos Exposure. Presentations for the Environmental Health and Safety Division, Georgia Tech Research Institute, Atlanta, Georgia, Asbestos Training Programs: Supervision of Asbestos Abatement Projects, 1987, 1988.

Dodson, R.F.: Lecturer -- Health Effects of Asbestos Exposure. Presentations for the Environmental Institute, Marietta, Georgia, Asbestos Training Programs: Asbestos in Buildings: Abatement Project Supervision, 1988.

Dodson, R.F.: Lecturer -- Health Effects of Asbestos Exposure. Presentations for International Association of Heat and Frost Insulators and Asbestos Workers, Houston, Texas, Asbestos Training Programs: Asbestos Abatement Seminar for Instructors, 1988.

Dodson, R.F.: Lecturer -- Health Effects of Asbestos Exposure. Presentations for the Environmental Research Institute, Inc., Tyler, Texas, and The University of Texas Health Center at Tyler, Asbestos Training Programs: Polarized Light Microscopy Course, 1988.

Dodson, R.F.: Lecturer -- Health Effects of Asbestos Exposure. Presentations for The University of Texas Health Center at Tyler Occupational and Environmental Training Programs, 1990-Present.

1. An Interdisciplinary Training Program for Practices and Procedures of Asbestos Abatement for Supervisors and Contractors.

2. An Interdisciplinary Training Program for Building Inspector for Asbestos Abatement Projects.

3. NIOSH 582 7400 Method - ORM (Air Sampling Course).

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Dodson, R.F.: S-collindine as a buffering system for glutaraldehyde perfusion of the central nervous system. J Neuropathology Exp Neurol 30:714-722, 1971.

Dodson, R.F.: Ultrastructural alterations of the medulla oblongata tissue following acute whole-body gamma irradiation. Acta Neuropathology 17: 353-362, 1971.

Garcia, J.H., Robinson, L., Dodson, R.F., and Velayas, E.: Electron microscopy of human skeletal muscle in health and disease (myositis). Amer J Pathol, February 1971 (abstract).

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Dodson, R.F. and Cheung, L.W.: Perivascular obligodendrocytes in the striatum of the squirrel monkey. J Neurol Sci 17:237-244, 1972.

Dodson, R.F., Hashi, K., and Meyer, J.S.: The effect of glycerol and intracarotid phenoxybenzamine after experimental subarachnoid hemorrhage: an ultrastructural study. Acta Neuropathol 24:1-11, 1973.

Dodson, R.F., and Wood, J.G.: Concentric membranous bodies in the central nervous system. Cytobios 7:61-69, 1973.

Dodson, R.F.: Electron Microscopy of microvascular pericytes in the brain. Cytobios 7:183-188, 1973.

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Exhibit Exhibits 1-19 to Dodson Declaration Page 37 of 61

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Exhibit 2

INDEX

No.	Date	Document
01	01-25-73	J&J Memo to Dr. Goodman, Dr. Rolle & W. Ashton RE: Talc/Asbestos No Advantage of looking into concentration method further
02	05-16-73	J&J Report-Proposed Specs for Analyzing Talc for Asbestos Tremolite in Vermont-Pooley Method
03	06-04-73	J&J Memo to Dr. Rolle from Shelley RE: Concern about tremolite in Vermont Talc
04	06-04-73	J&J Memo to Ashton, Dr. Fuller, Dr. Goudle, Dr. Hildick-Smith, Dr. Lord, Dr. Nashed and Dr. Patterson RE: Concern over concentrated talc test methods
05	06-06-73	J&J Memo to Mortimer, Miller, Johnston & Stolzer from Petterson RE: Pooley found actinolite using the concentration technique
06	09-13-73	Pooley Report found tremolite actinolite in Italian mine and Vermont mine
07	10-23-73	J&J Memo to Dr. Petterson from Ashton RE: CSM and JM looking too closely at concentration of asbestos minerals from our talc systems
08	11-19-73	Pooley concentration method, asbestos in cosmetic talc negligible
09	11-21-73	Ltr to J&J from Dr. Nashed RE: JJ agrees with Pooley concentration method
10	12-27-73	Colorado School of Mines Research Institute – A Procedure to Examine Talc for Presence of Chrysotile & Tremolite-Actinolite Fibers Repot to J&J explaining concentration method
11	March 1974	Memo to Windsor Minerals from Dept of Earth Sciences, Dartmouth College RE: Analysis of Talc Products & Ores for Asbestiform Amphiboles
12	March 1974	Special Talc Studies Monthly Report for March 1974 J&J had their own concentration method
13	03-11-74	J&J Memo to Dr. Schelz and Dr. Rolle from Carpe RE: Methods of Concentration of Asbestos in Talc-Project #0503-00
14	April 1974	Special Talc Studies Monthly Report for April 1974 Pooleys concentration method quartz in JJBP
15	02-18-75	J&J ltr to Dr. Rolle RE: deliberately not including concentration technique
16	02-28-75	J&J memo to Sloan from Rolle RE: Review of CTFA Methodology for the Detection of Asbestos in Talc, as well as, Comments on TPF Methodology
17	11-24-76	J&J Memo to George Lee from Ashton RE: FDA Concentration Technique Disturbing
18	10-17-80	Procedure for Identification and Quantification of Amphibole Particles in J-J Talcs Using Optical Mineralogical Microscopy (JJ allowed amphibole greater than 5 um in their talc samples
19	1991	Amphibole Content of Cosmetic & Pharmaceutical Talcs by A. M. Blount
20	01-27-93	Ltr to Joselyn of Cahill, Gordon & Reindel from Stewart at RJ Lee Group- RE: Talc Sample Analysis- RJ Lee Group Project # LBH211626 (used Blount method)
21	09-01-14	International Standard- Air quality-Bulk materials- Part 2: Quantitative determination of asbestos by gravimetric and microscopical methods

EXHIBIT 2

Exhibit 3

Index
Hayes v. Colgate , et al.

A: Alice Blount Article &Correspondence

1. Amphibole Content of Cosmetic and Pharmaceutical Talcs (1991) Full paper

2. Amphibole Content of Cosmetic and Pharmaceutical Talcs (1991)

3. Correspondence to Raymond Hatcher from Dr. Blount (04-23-98)

B: J&J Memos RE: Health Impacts from Cosmetic Talc

1. Memo from Steinberg to Dr. G. Hildick-Smith, RE: Johnson's Baby Powder Talc Aspiration (06-17-66)

2. Memo from Walter Newman, RE: Johnson's Baby Powder, Project No. 0519.00 Claim Support (02/10/74)

3. Talc: A Basic Review Professional Information for Physicians and Nurses, Prepared for Dr. William Waggoner, Mgr. Regulatory Affairs Johnson & Johnson by J.L. Shapiro Associates (06-10-76)

C: J&J Attempts to Remove Asbestos

1. Correspondence to William Ashton, J&J Research Division from Earl Smith, Desert Minerals, Inc. Products Marketing Manager, RE: Discussion regarding proposed FDA regulations for talc (02-08-73)

2. Correspondence to Earl Smith, Celite Division of Johns-Manville Corp from W.T. Caneer, Projects Manager Colorado School of Mines Research Institute, RE: Question regarding physical removal of 100% tremolite which may occur with talc (02-20-73)

3. Correspondence to Ian Sloan, J&J from T.H. Shelley, Ph.D., Director Central Research Laboratories, RE: Professor Pooley's process to remove tremolite from talc (02-20-73)

4. J&J Memo to G. Lee from W.H. Ashton, RE: Notes on Visit to Denver Area March 25 thru March 27, 1974 (04-04-74)

5. J&J Memo to Dr. D.R. Petterson from G. Lee, RE: Status-New Flotation Methods Project No. 1133.02 (06-20-74)

EXHIBIT 3

D. J&J J4-1 and TM 7024 Methods

1. Report of CTFA Talc Subcommittee on Method to Detect Chrysotile and Tremolite in Talc (12-10-73)

2. J&J Memo to File from G. Hildick-Smith, RE: Talc/Asbestos Meeting with Commissioner Schmidt, FDA, January 16, 1974(01-18-74)

3. Correspondence to Heinz Hiermann, FDA from Norman Estrin, PH.D., CTFA, RE: current status of talc (03-15-76)

4. CTFA Talc Subcommittee Minutes by Norman Estrin, Ph.D., VP-Science (03-31-76)

5. CTFA Minutes-CTFA Task Force on Round Robin Testing of Consumer Talcum Products for Asbestiform Amphibole Minerals by Mort Westman, Director of Cosmetic Sciences (06-13-77)

6. CTFA Minutes- Talk Task Force on Round Robin Testing by Norman Estrin, Ph.D., VP-Science (12-06-77)

7. Correspondence to Charles Haynes, CTFA from John Schelz, CTFA Task Force on Round Robin Testing of Consumer Talcum Products (03-01-78)

8. Stipulation of Dismissal as to Windsor Minerals, with attached affidavit of Roger Miller dated July 3, 1987 (07-23-87)

9. Correspondence to Michael Keener, Quality Control Mgr, Cyprus Windsor Minerals Corporation from E. Kent Sprague, Electron Microscopist, McCrone, RE: talc samples for asbestos analysis (11-26-90)

10. J&J Memo to D. Jones Regina Gallagher, Principal Scientist, RE-Summary of Raw Material & Finished Product Testing for Baby Powder Talc (01-10-94)

11. J&J Consumer Companies Worldwide Specification- Analysis of Powdered Talc for Asbestiform Minerals by Transmission Electron Microscopy (8-21-95)

12. Memo to Tim Hicks from Julie Pier, RE: Explanation of "detection limit" on J&J TEM analysis of Grade 66 (12-05-96)

13. J&J Material Specification, RE: Luzenac America, Inc., Windsor Grade 66 and 96 Talc (11-15-01)

14. Email to Julie Pier from Gary Tomaino with attached Proposed presentations for 2011 Johnson Conference on asbestos (10-15-10)

E. J&J Testing Methods

1. Memo to W. H. Ashton & Others from F. Robert Rolle, Ph.D., RE: Meeting with FDA (9-21-72) to discuss chrysotile asbestos in Shower to Shower Powder-Project No. 503 (09-26-72)

2. Memo to D.D. Johnston from D.R. Petterson, RE: Windsor Minerals and Talc (04-26-73)

3. Memo to J. Mullen from W. Nashed, RE: Proposal will have no impact on our talc (10-23-73)

4. Memo to Dr. E.R. L. Gaughran and Dr. T.H. Shelly from A.J. Goudie, RE: Purchase of a Transmission Electron Microscope Plus Attachments (01-03-74)

5. Memo to Windsor Minerals Inc., Windsor, Vermont 05089 from R.C. Reynolds Jr., Dept of Earth Sciences, Dartmouth College, Hanover, New Hampshire 03755, RE: Analysis of Talc Products and Ores for Asbestiform Amphiboles (March 1974)

6. Memo to M.F. Warner from G.J. Gill, RE: Pfizer Company, Easton, Pennsylvania-Asbestos in Talc Analysis (06-24-77)

7. Memo to M. Cox from R.J. Zanenski, RE: Due Dilligence of Windsor Minerals Quality Control Program (10-05-88)

8. Letter to John C. O'Shaughnesay of J&J from James Guay, Manager of Purchasing of Luzenac America, RE: Reply to letter of 10-4-94 in reference to Ritter v. Cyprus, et al. (10-17-94)

9. Email to Donna Dennis from Rich Zazenski, RE: Talc Specification (06-07-01)

10. Memo to Ed McCarthy (RTM and Shripal Sharma (RTM) from Julie Pier, RE: USP Method (06-15-11)

11. Rio Tinto Minerals Issue Briefing-talc quality assurance letters (no date)

F. Presence of Asbestos in Vermont Talc

1. J&J Memo W. Ashton, RE: Metropolitan Talc Lot G 716, Preliminary Evaluation (11-01-67)

2. Correspondence to Dr. A.J. Goudie of J&J from Ian Stewart, Manager, Electron Optics, McCrone Associates, RE: McCrone Study being redone (10-27-72)

3. Examination of Johnson and Johnson Baby Powder to Dr. A.J. Goudie of J&J from Ian Stewart of McCrone. "Do not use this report, replaced by another version" (10-27-72)

4. New Reagent Systems-Plant Trial at Windsor Minerals, Inc. by Vernon Zeitz, Manager Research and Development, Windsor Minerals, Inc. (05-14-74)

5. Laboratory Report Cyprus Industrial Minerals Company, Report: 7690, 1790-79, KEY: Windsor Minerals/Illinois EPA Subject: Electron Microscopy of Windsor Minerals Talc Samples (02-24-75)

6. Correspondence to Vern Zeitz, Windsor Mineral Company from Gene R. Grieger, Walter McCrone Associates, RE: samples using electron microscopy and selected area electron diffraction to determine extent of amphiboles or serpentine contamination in these two groups of samples (07-01-75)

7. Correspondence to Vernon Zeitz of Windsor Minerals Company from Gene R. Grieger, Walter McCrone Associates, RE: Supplement report of July 1, 1974 on series of talc ore samples (11-05-75)

8. Vermont Due Diligence: Product Quality and Quality Control, Sampled and Inspected by: R.J. Zaneski, K.W. Olson, M.L. Clark. Analysis performed by: Product Certification Lab-Cartersville, GA; Skyline Laboratories-Denver, CO; and Aquatec Environmental-Burlington, VT; This Report contains Executive Summary, Summary by Location and Appendix of all data and analyses (04-15-88)

9. Interoffice Correspondence to Kerstetter, Toll, Lawson, Paulsen and Moreau from R.C. Munro, RE: Cyprus Ore Reserves-Arsenic & Tremolite (03-25-92)

10. Luzenac America Memo to Philip Moreau from Michael Clark, RE: Actinolite for Toxicology Study, Project No: 93-077b (11-02-93)

11. Luzenac America Memo to David Crouse from Julie Pier, RE: Analysis of Fibrous Material From Argonaut Waste Rock (05-23-02)

12. Quantitative Analysis Report Asbestos in Bulk Material, Date Reported 01-05-04

13. Memo to Dr. Goudie from W. Nashed, RE: Tremolite was mistakenly identified in view of similarity to Na Sesquicitrate (No Date)

G: Hamm Mine & Hammondsville Mine

1. J&J Memo to Mr. R.C. Stites from J.T. Dettre, RE: Windsor Minerals Board Meeting (11-08-73)

2. J&J Memo to Dr. J.R. Marshall from Alan M. Marks, RE: Johnson's Baby Powder-Support; Argonaut Talc-Alternate Supply Project No. 2636.01 (07-16-76)

3. Letter to Carol Wilkes of J&J from Tim Hicks of Luzenac America, RE: Bacteria count for Grade 66 silo #4 (01-05-96)

4. Luzenac America Inc. Memo to Nancy Oyer, Lyzenac Technical Center from Joan R. Johnson, Quality Assurance Lab Supervisor, RE: Samples for Product Certification by X-RD, TM and SEM from West Windsor Lab and Columbia Mill Lab. Note: Mixture of Hamm-Argonaut Ores started 9/18/95. Please check for serpentine and send West Windsor Lab conformation that serpentine is non fibrous form (01-09-96)

5. Memo to J.R. Cowans, W. Windsor from E.F. McCarthy/C.J. DeMarco, RE: Pilot Flotation Study on Argonaut Ore at W. Windsor (01-31-96)

6. J&J Material Specification, RE: Luzenac America, Inc., Windsor Grade 66 Talc (Issue date: 03-09-00)

7. IV. Brief History of Operations (No date)

8. Argonaut Fact Sheet (No Date)

9. Cyprus Ore Reserve Evaluation Preliminary Summary by R.C. Munro (No date)

Exhibit 4

cc: Dr. R. A. Fuller
Dr. W. Nashed
Mr. R. J. Mortimer to Mr. R. N. Miller
Dr. T. H. Shelley
Dr. R. L. Sundberg

May 14, 1971

Assay of Talc in BABY POWDER

Dr. G. Hildick-Smith

The attached letter shows the particle size-shape consists of a production batch of our product produced in December, 1970. It is an aliquot of that which I gave to Dr. A. Langer at Mt. Sinai Hospital. As you know I had this done to see what type of data we can expect from independent experts, as we had agreed.

You will note that 99% of our product is platy when assayed on a surface particle area basis. On the other hand 87% is platy when observed on a particle count basis. It is more usual to run a particle count assay in the business.

We consider the free non-talc needles but a trace, both on a count and area basis. Those particles are tremolite.

However, if such an assay were to be run by microscopists who are not aware of the differences between fibrous talc, broken talc plates, and tremolite we can then expect them to report about 5.5% needles by count and at least 0.5% needles by area.

You will note we are now picking up observable amounts of quartz in this particular batch. I have no explanation for that at present.

In addition to the attached I ran an X-ray diffractograph on that batch. It shows these minerals are present:
talc, mica, chlorite, tremolite-actinolite, and magnesite.

I am proceeding to investigate samples of talc used during the past year as we discussed on Wednesday.

W. H. Ashton

rap
Attachment

EXHIBIT 4

COLORADO SCHOOL OF MINES RESEARCH INSTITUTE
P.O. BOX 112
GOLDEN, COLORADO 80401

May 10, 1971

REFER TO
390517

Mr. Wm. Ashton
Johnson & Johnson
Research Center
Research and Development
New Brunswick, New Jersey 08901

Dear Mr. Ashton:

Following are the results of the point count analysis on sample 344L baby powder:

Free Particles

	No. of Grains	%	Cum sq mm	%	Avg sq mm
Platy talc and/or chlorite (with inclusions)	434	87.32	1.200355	99.10	0.002765
Free diamond shaped particles	5	1.01	0.000148	0.01	0.000030
Free carbonates	7	1.41	0.001857	0.15	0.000265
Free quartz	8	1.61	0.003236	0.27	0.000404
Free talc shards	14	2.82	0.004945	0.41	0.000353
Free nontalc needles	3	0.60	0.000081	0.01	0.000027
Free talc needles	11	2.21	0.000242	0.02	0.000022
Free other (0.0016 x 0.0016 mm)	14	2.82	0.000186	0.01	0.000013
Dark opaque	1	0.20	0.000215	0.02	0.000215
Total	497	100.00	1.211265	100.00	

Mr. Wm. Ashton
Johnson & Johnson
CSMRI Project No. 390517
Page 2

Inclusions in Talc and/or Chlorite Plates

	No. of Grains	%	Cum sq mm	%	Avg sq mm
Other (0.0016 x 0.0016 mm)	1,384	85.28	0.003719	27.58	0.000003
Talc needles	75	4.62	0.002045	15.16	0.000027
Quartz	52	3.20	0.001639	12.15	0.000032
Diamond shaped particles	56	3.45	0.000853	6.33	0.000015
Talc shards	11	0.68	0.002894	21.46	0.000263
Carbonates	9	0.55	0.000814	6.04	0.000091
Nontalc needles	36	2.22	0.001521	11.28	0.000042
Total	1,623	100.00	0.013485	100.00	

Platy talc and/or chlorite	1.200355	= total platy talc and/or chlorite with inclusions
Total inclusions	(-) 0.013485	= total sq mm of inclusions
	1.186870	= total platy talc and/or chlorite minus inclusions

On a cumulative sq mm basis, the talc and/or chlorite plates examined contained a total of 1.12% inclusions.

Following are explanations of the terms used in the above data tables.

1. The "inclusions" noted in the talc and/or chlorite plates were nonopaque particles. No dark opaque particles were noted as inclusions.

2. The "diamond shaped particles" were nonopaque with a refractive index above the 1.600 oil used. Measurments of the internal angles of the crystals were close to 90° in all cases indicating they could be some variety of pyroxene.

3. The "carbonates" noted may include magnesite, dolomite or calcite.

Mr. Wm. Ashton
Johnson & Johnson
CSMRI Project No. 390517
Page 3

4. The "quartz" noted was identified using interference figures where possible. Where not possible, due to size, concoidal fracture and interference colors were used for identification.

5. The "talc shards" noted were thin pieces that could clearly be defined as being pieces broken off of plates, etc. This entailed very detailed study of each piece to determine if lamination or ragged edges were present.

6. The "nontalc" needles were identified as such if it was readily observable the particles were true needles and if they had extinction angles greater than 3° (usually 10-20°).

7. The "talc" needles were identified as such if it was readily observable the particles were true needles and if they had extinction angles of 3° or less.

8. The "other (0.0016 x 0.0016 mm)" particles were nonopaque particles that had had refractive indices greater than the 1.600 oil used. These particles were so small no other optical data could be procured from them.

The point count was made at 500X.

If you have any questions regarding this data, please call me.

Sincerely,

M. G. Pattengill
Project Engineer

/laj

Exhibit 5

LABORATORY OF SURGICAL PATHOLOGY

STANFORD UNIVERSITY MEDICAL CENTER
300 PASTEUR DRIVE, ROOM H-2110, STANFORD, CALIFORNIA 94305
TEL #: (650) 723-7211 FAX #: (650) 725-7409
Gerald J. Berry, MD Director of Anatomic Pathology
Ann K. Folkins, MD Director of Surgical Pathology

Patient: **HERNANDEZ-VALDEZ, ANTHONY MICHAEL**

Pathology No: **SHS-22-07213**

Med. Rec. No.: **36945558**
Sex: M Age: 23
Date of Birth: 9/23/1998
Account No.: 131325214011

Date of Procedure: 2/17/2022
Date Received: 2/17/2022 6:00:00 AM

Physician(s):
JACK H BOYD, M.D.
STANFORD HEALTH CARE
500 PASTEUR DR
STANFORD, CA 94304

CLINICAL HISTORY: 23 year old male with mesothelioma of pericardium ; Per EPIC: 23-year-old male with pericardial mesothelioma. He has been offered palliative pericardiectomy for tumor debulking with the hope of relieving his shortness of breath as well as PleurX catheter placement.

OPERATION: 1. Pericardiectomy (performed by Dr. Boyd) 2. Bilateral PleurX catheters 3. Resection of mediastinal mass and thymectomy

OPERATIVE FINDINGS: 1. Large bilateral chylothoraces. 2. Diffuse tumor involvement of the pericardium with areas of invasion into the myocardium

CLINICAL DIAGNOSIS: 1. Pericardial mesothelioma. 2. Bilateral pleural effusions, chylothoraces. 3. Pericardial constriction.

GROSS DESCRIPTION: Two specimens are received labeled with the patient's name and medical record number.

The first specimen labeled "thymus pericardial fat" is received in formalin and consists of multiple fragments of tan-yellow tissue, aggregate 11.0 x 9.5 x 3.0 cm. A photo is taken. Specimen is sectioned to reveal yellow lobulated cut surfaces with areas of white nodularity, up to 1.8 cm in greatest dimension. Representative sections, including candidate normal thymus, are submitted in A1-A4.

The second specimen labeled "pericardium tumor" is received in formalin and consists of multiple roughened tan-brown tissue fragments, aggregate 7.5 x 7.0 x 2.0 cm. Sectioning reveals nodular tan-white cut surfaces. Representative sections are submitted in B1-B4. Mirbegian (2/21/2022)

I have reviewed the specimen and agree with the interpretation above. GERALD J. BERRY, M.D.
Electronically signed 2/23/2022 1:21 PM

Christina S. Kong, M.D. – Medical Director

EXHIBIT 5

Exhibit 16

Marc E. Wolin, Esq.
mwolin@saiber.com
John M. August, Esq.
jaugust@saiber.com
SAIBER LLC
18 Columbia Turnpike, Suite 200
Florham Park, NJ 07932
Tel: (973) 622-8401

-and-

Joseph D. Satterley, Esq.
jsatterley@kazanlaw.com
Denyse F. Clancy, Esq.
dclancy@kazanlaw.com
KAZAN, McCLAIN, SATTERLEY & GREENWOOD
A Professional Law Corporation
Jack London Market
55 Harrison Street, Suite 400
Oakland, CA 94607
Tel: (510) 302-1000

Counsel for Claimant Anthony Hernandez Valadez

IN THE UNITED STATES BANKRUPTCY COURT
FOR THE DISTRICT OF NEW JERSEY

In re:	:	Chapter 11
LTL MANAGEMENT LLC,	:	Case No. 21-30589
Debtor.	:	

CLAIMANT ANTHONY HERNANDEZ VALADEZ'S STATUS UPDATE

The law firm of Kazan, McClain, Satterley & Greenwood, A Professional Law Corporation, and local counsel Saiber LLC, on behalf of personal-injury claimant Anthony Hernandez Valadez, through his undersigned counsel, hereby submit this Status Update to apprise this Court of further developments in Mr. Valadez's case and to assist in the estimation

EXHIBIT 16

of damages for mesothelioma plaintiffs. In support of this Status Update, Mr. Valadez respectfully represents as follows:

BACKGROUND

1. Mr. Valadez recently celebrated his 24th birthday on September 23, 2022. He is suffering from pericardial mesothelioma, a terminal and asbestos-caused cancer.

2. On June 14, 2022, this Court granted Mr. Valadez limited relief by allowing him to file a complaint in the Superior Court of California, County of Alameda. The next day, Mr. Valadez filed his complaint. He alleged that Johnson & Johnson and the retailers who sold its Johnson's Baby Powder talc exposed him to asbestiform fibers and caused his mesothelioma.

3. On August 8, 2022, this Court granted Mr. Valadez limited relief from the automatic stay and preliminary injunction order "to conduct such discovery as necessary to preserve evidence pertinent to Mr. Valadez's claim that may otherwise be lost or destroyed, including, but not limited to Mr. Valadez's pathology materials." [Main Dkt. 2836 at 3.]

FURTHER DEVELOPMENTS IN MR. VALADEZ'S CASE AND HEALTH

I. Mr. Valadez completed his deposition.

4. Over four days in mid-September 2022, Mr. Valadez testified about his life, use of Johnson's Baby Powder talc, and medical condition. [Valadez Decl. at ¶ 3.] As to Johnson's Baby Powder, Mr. Valadez testified that he used the product daily, sometimes several times, as part of his hygiene routine. [*Id.*] Applying baby powder created visible dust that Mr. Valadez breathed. [*Id.*]

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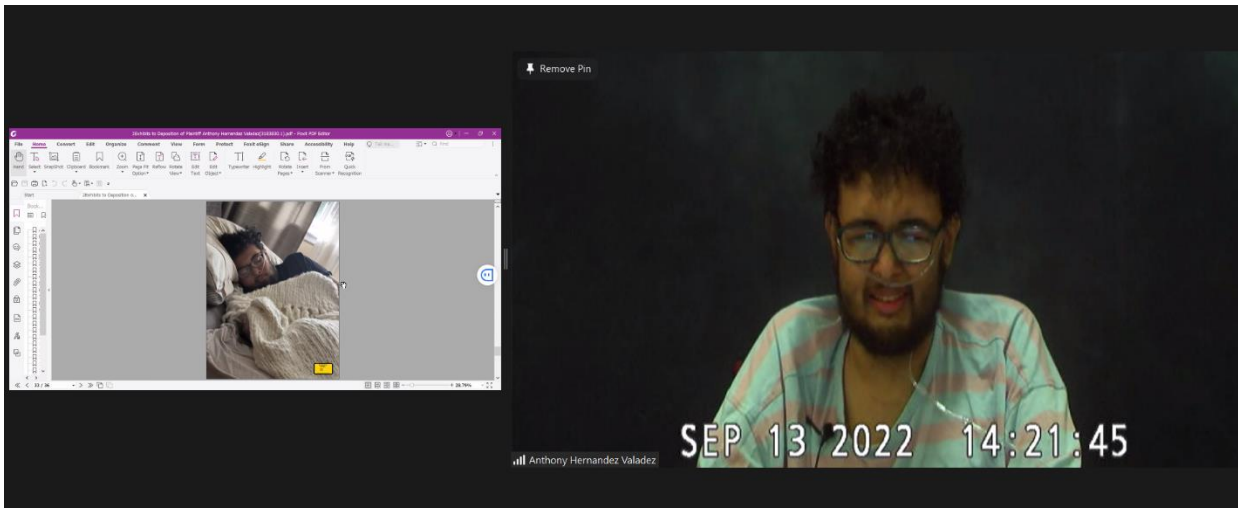
5. Throughout his four days of deposition, Mr. Valadez constantly experienced body pain, headaches, stress, and anxiety. [Valadez Decl. at ¶ 4.] Also, Mr. Valadez used supplemental oxygen because of his breathing difficulties. [*Id.*]



II. Mr. Valadez's disease is advancing.

6. Thus far, Mr. Valadez has undergone five cycles of immunotherapy. [Valadez Decl. at ¶ 5.] This treatment does not cure mesothelioma. [*See* Dr. Roy Decl., Main Dkt. 2469-3 at 5-6.] Instead, Mr. Valadez is undergoing immunotherapy treatment to slow the progression of his mesothelioma. [*Id.*]

7. However, Mr. Valadez's recent CT scan, dated October 7, 2022, shows that his disease is progressing despite immunotherapy. [Valadez Medical Records, Exh. 2 hereto at 2-3.] The results reveal "enlarging pericardial nodules" in his cardiovascular area and "enlarged pleural nodules" in the lung parenchyma, pleura, and airways. [*Id.*]

8. Mr. Valadez still has significant anxiety and depression. [Valadez Decl. at ¶ 6.] Merely talking about his mental state triggers Mr. Valadez's emotions. [*Id.*] Recently, his anxiety and stress were exacerbated when Mr. Valadez experienced temporary blindness after undergoing a cycle of immunotherapy. [*Id.* at ¶ 5.]

9. Mr. Valadez's disease and any treatments he receives have significantly impacted his physical abilities. [Valadez Decl. at ¶ 7.] He experiences nausea/vomiting, poor appetite, chest pain and tightness, breathing difficulties, discomfort, fatigue, and body pain. [*Id.*]

III. Johnson's Baby Powder talc exposed Mr. Valadez to asbestiform fibers and played a causative role in his mesothelioma.

10. Dr. Richard Kradin is a pulmonologist and pathologist. [Kradin Report, Exh. 1 hereto at 1.] He has specialized in pulmonary disease for over 38 years and is board certified in Internal Medicine, Anatomic Pathology, and Pulmonary Medicine. [*Id.*] He is an Associate Physician and Pathologist (Honorary) at Massachusetts General Hospital and an Associate Professor of Pathology and Associate Professor of Medicine Emeritus at Harvard Medical School. [*Id.*]

11. Dr. Kradin has reviewed Mr. Valadez's diagnostic pathology materials and confirmed that Mr. Valadez has mesothelioma. [Dr. Kradin Report, Exh. 1 hereto at 20.] As shown in prior filings, Johnson & Johnson knew that there was asbestos in its talc baby powder. [Valadez's Motion to Lift Stay, Main Dkt. 2348-1 at 13 and evidence cited therein.] Similarly,

other labs have documented the presence of asbestos and asbestiform fibers in Johnson's Baby Powder. [*Id.* at 13-14 and evidence cited therein.]

12. Given Mr. Valadez's "history of exposure to cosmetic talc," Dr. Kradin opines, "to a reasonable degree of medical probability that asbestos played a causative role in Mr. Valadez's mesothelioma." [Dr. Kradin Report, Exh. 1 hereto at 21.]

IV. Mr. Valadez has strong evidence against Johnson & Johnson, and any estimation of his damages is very significant.

13. Mr. Valadez's case demonstrates that he has substantial evidence to support his claims against Johnson & Johnson. Dr. Kradin, Mr. Valadez's two treating doctors at Stanford University, and Stanford University medical professor Dr. Dean Felsher have opined that Mr. Valadez's decades of exposure to asbestiform fibers from his and others' use of Johnson's Baby Powder increased his risk of developing mesothelioma. [Dr. Backhus Decl., Main Dkt. 2348-13, at 7-8; Dr. Felsher Decl., Main Dkt. 2348-15, at 5-9; Dr. Roy Decl., Main Dkt. 2469-3, at 7-8; Dr. Kradin Report, Exh. 1 hereto at 20-21.]

14. Given the strength of Mr. Valadez's evidence against Johnson & Johnson, the damages here are huge. Any estimation of damages in malignant mesothelioma cases, such as Mr. Valadez's, is very significant. Indeed, Mr. Valadez's economic damages alone are nearly $3.7 million. [Robert Johnson Decl., Main Dkt. 2348-23, at 3 and Exh. B thereto.] And that is just a tiny portion of the total damages which, under California law, includes past and future physical pain, mental suffering, loss of enjoyment of life, disfigurement, physical impairment, inconvenience, grief, anxiety, humiliation, and emotional distress. *Bigler-Engler v. Breg, Inc*., 7 Cal.App.5th 276, 300 (Cal. App. 4th Dist. 2017).

15. Accordingly, Mr. Valadez has viable claims against Johnson & Johnson, and any estimation of his damages should be in the millions of dollars.

CONCLUSION

16. Since October 14, 2021, over 14 months, Johnson & Johnson has not negotiated with any mesothelioma victims to compensate them for the horrible damage it has inflicted from its negligent and reckless misconduct of knowingly marketing a carcinogenic product. This Court should lift the automatic stay so that mesothelioma victims like Mr. Valadez can regain their Seventh Amendment rights to a jury trial during their lifetime.

Respectfully submitted:

KAZAN, McCLAIN, SATTERLEY & GREENWOOD, A Professional Law Corporation

- and -

SAIBER LLC
Counsel for Claimant Audra Johnson

By: /s/ John M. August
JOHN M. AUGUST

DATED: December 15, 2022

Exhibit 1

KRADIN CONSULTING

Richard L. Kradin, M.D., DTM&H
15 Constitution Drive (Suite 1A)
Bedford, NH 03110

Tel: (781)367-3641
Email:richardkradin@gmail.com

Honorary Pathologist and Physician
Massachusetts General Hospital

Associate Professor Emeritus of Medicine and Pathology
Harvard Medical School

Adjunct Faculty, Hadassah Hospital, Jerusalem IL

December 6, 2022

Ian Rivamonte
Kazan, McClain, Satterley & Greenwood
Jack London Market
55 Harrison Street, Suite 400
Oakland, CA 94607
Ph: 510 302-1000
Fax 510 835-4913

RE: ANTHONY H. VALADEZ DOB:09-23-1998

My name is Richard L. Kradin, M.D., D.T.M & H, and I am over 18 years of age and issue this report based on my personal knowledge. The following opinions and statements are made with a reasonable degree of medical probability based on my knowledge, experience, expertise and training, on generally accepted medical and scientific principles, and on peer-reviewed and published literature described herein. The methodology and basis for these opinions are not novel, are generally accepted in the medical and scientific community, and have been subjected to peer-review and publication. While I may not agree with every opinion in the documents cited herein, the materials discussed below are a reliable basis for my opinions in this case, and they are the types of materials that physicians normally rely upon to formulate our opinions in the everyday practice of medicine, and outside of the litigation context.

QUALIFICATIONS

I am a pulmonologist and pathologist licensed to practice in Massachusetts and Florida. I have specialized in pulmonary disease for over 38 years. I am board certified in Internal Medicine, Anatomic Pathology, and Pulmonary Medicine. My areas of sub-specialization include pulmonary pathology and autopsy pathology.

I am an Associate Physician and Associate Pathologist (Honorary) at Massachusetts General Hospital, and an Associate Professor of Pathology and Associate Professor of Medicine Emeritus at Harvard Medical School. In addition to my M.D., I have an advanced degree in chemical physics and have studied epidemiology at the London School of Hygiene.

Since the 1980s, I have had a special interest in the clinical features and pathology of asbestos-related diseases. I direct postgraduate work on asbestos-related diseases at Harvard Medical School and am the Principal Investigator on several research projects, including one involving asbestosis. I am the Director of a yearly seminar on asbestos-related pulmonary disease and co-director of a yearly seminar on thoracic pathology, both through the Harvard Medical School. I routinely read the literature concerning fiber release from various asbestos containing products, including insulation products, and the effects on the

human body. I am also familiar with what has been published regarding the level of asbestos present in the ambient air.

I have authored approximately 200 articles, including those related to asbestos and its diseases. I have authored three textbooks of pathology. I have conducted research on the immunological defenses of the lung to soluble and particulate agents and have been the Primary Investigator for National Institutes of Health-sponsored clinical trials regarding the immunological treatment of lung and other cancers.

In addition to research and teaching, I have personally cared for patients with both benign and malignant asbestos-related diseases and have frequently had the opportunity to review pathology specimens from patients with asbestos-related diseases at Massachusetts General Hospital as well as cases sent to me from outside sources for my opinion based on my established expertise with the diagnoses of these disorders. I have personally reviewed hundreds of biopsies of asbestos-related malignancies, including malignant mesothelioma, and I have performed numerous autopsies in patients with asbestos-related diseases.

Since the 1980s I have testified in court on many occasions as an expert on asbestos-related diseases, including in personal injury trials in various states, including the States of Louisiana, Texas, California, Pennsylvania, Maryland, as well as others. I have been an expert witness in asbestos litigation on behalf of the U.S. Government. My qualifications are more fully described in my Curriculum Vitae.

GENERALLY ACCEPTED PRINCIPLES REGARDING MESOTHELIOMA

1. ASBESTOS CAUSES MESOTHELIOMA

Mesothelioma occurs when asbestos fibers cause genetic errors in mesothelial cells within the lining of the chest, abdomen, or around the heart – that is, the pleural, peritoneal, and pericardial membranes. Multiple genetic errors must take place before the cancer develops. The damage to DNA can occur as soon as the fibers reach the target cells. In the case of mesothelioma, within hours or days after exposure, asbestos fibers that are transported to the pleura are taken up by the mesothelial cells and rapidly cause genetic errors and other damage that leads to genetic errors. Inhalation of asbestos can and does also increase cell division and other biological responses that promote the development of cancers and/or reduce the effectiveness of the body's defense mechanisms for fighting the development of cancer. By the time an individual is diagnosed with mesothelioma, multiple epithelial or mesothelial cells have accumulated a series of genetic errors from the asbestos fibers. Eventually, in persons who develop a mesothelioma, one of these multiple cells with multiple genetic errors escapes the body's defense mechanisms and replicates to form the mesothelioma.

It is generally recognized in the medical community that asbestos is a complete carcinogen, which means it can both initiate and promote cancer. Therefore, the persistent asbestos fibers and additional exposures after the initial exposure cannot be discounted in determining causation. Rather, the cumulative dose of asbestos causes mesothelioma through both direct and indirect mechanisms over the evolution of the cancer. (IARC Monograph on the Evaluation of Carcinogenic Risks to Humans 2012; 100C:219-309) It is generally recognized in the medical community that all types of asbestos fibers cause mesothelioma, which is discussed below in more detail.

2. MESOTHELIOMA IS A SIGNATURE DISEASE FOR ASBESTOS EXPOSURE

Mesothelioma is a rare malignancy. It is generally accepted in the medical community that asbestos causes mesothelioma and that the vast majority of mesotheliomas are caused by asbestos. For other diseases, such as lung cancer, asbestos is just one of many causes that often work in combination in the causation of cancer. A more complete discussion of the multi-factorial causes of lung cancer is beyond the scope of this report.

The causal relationship between exposure to asbestos and mesothelioma is so firmly established in the scientific literature that mesothelioma is considered a "sentinel," "signature" or "signal" tumor for asbestos exposure. This means that the presence of mesothelioma generally "signals" prior asbestos exposure; this is true even for individuals who cannot recall the exposures — because those exposures occurred decades ago, were unknown to the individual at the time of exposure, or for some other reason. Indeed, many individuals are not always aware that they have a history of exposure to asbestos. (Leigh et al. 2002."Past exposure is not always recognized as such and this is more likely to be the case in females. Indeed, even absence of fibers in the lungs does not negate exposure as fibers may have initiated mesothelioma and then been cleared before death." pp. 194, 198)

3. MESOTHELIOMA IS A DOSE-RESPONSE DISEASE

Mesothelioma is a dose-response disease, which means that the more someone is exposed to asbestos, the greater their *risk* for developing mesothelioma. If a person is exposed to fewer asbestos fibers, then fewer fibers can make their way to the pleura or peritoneum. On the other hand, if a person is exposed to larger numbers of asbestos fibers, more will likely make their way to the pleura or peritoneum. This is the nature of the dose-response relationship between asbestos exposure and mesothelioma: the more asbestos exposure an individual has, the greater his or her chance of developing mesothelioma. However, for an individual who develops mesothelioma and has had multiple exposures to asbestos (for example, at different jobs or to different products), it does not follow that the shorter or briefer exposures did not play a role in causing the disease.

It is generally accepted in the medical community that a physician need not, and indeed cannot, identify the specific exposure or asbestos fibers that directly "caused" the mesothelioma – that is, which fibers caused the genetic errors that transform a non-cancerous cell into a cancer cell. Trying to identify which exposures to asbestos began the malignant process for mesothelioma is like pointing to the pack of cigarettes or the specific cigarette or inhalations that cause a smoker's lung cancer – that is impossible and makes no sense scientifically. As a practical matter, a worker is never exposed to a single or small numbers of asbestos fibers; rather occupational and para-occupational exposures to asbestos invariably involve fibers released and inhaled in enormously high numbers.

4. ALL FORMS OF ASBESTOS CAN CAUSE ALL ASBESTOS-RELATED DISEASES, INCLUDING MESOTHELIOMA

There is overwhelming, generally accepted evidence that inhalation of asbestos fibers of any type (chrysotile, amosite, crocidolite, tremolite, anthophyllite and actinolite), from any source or product, causes all asbestos-related diseases, including mesothelioma. Mesothelioma is a tumor of the serosal linings of the chest (the pleura), the abdomen (peritoneum), the heart (pericardium) and testes (tunica vaginalis). The cells of the serosal membranes surrounding the lungs, abdomen, heart and testes are essentially the same, at a cellular level, and react to asbestos in the same manner. All variants of diffuse

malignant mesothelioma, in any location of the body, can be caused by all forms of asbestos, including chrysotile. These generally accepted principles regarding asbestos disease causation, and the methodology behind them, are not new or novel in the medical and scientific community.

Various occupational epidemiology, registry and cases studies clearly link all types of asbestos, including chrysotile asbestos, to pleural and peritoneal mesothelioma. The following are examples of the many studies, texts and reports that support or form the basis for my opinions:

- Hein, et al., *Follow-up study of chrysotile textile workers: Cohort mortality and exposure response*, Occup. Environ. Med. 64:616-625 (2007).
- Everett, et al., *Occupational asbestos exposure among respiratory cancer patients in Lithuania*, Am. J. Ind. Med. 50:455-463 (2007).
- Mirabelli, et al., *Excess of mesotheliomas after exposure to chrysotile in Balangero, Italy*, Occup. Environ. Med. 65:815-819 (2008).
- Loomis, et al., *Lung cancer mortality and fiber exposures among North Carolina asbestos textile workers*, Occup. Environ. Med. 66:535-542 (2009).
- Nishikawa, et al., *Recent mortality from mesothelioma. Historical patterns of asbestos use and adoption of bans: A global assessment*, Environ. Health Perspect. 116:1675-1680 (2008).
- Silverstein, et al., *Developments in asbestos cancer risk assessment*, Am. J. Ind. Med. 52:850-858 (2009).
- Pira, et al., *Mortality from cancer and other causes in the Balangero cohort of chrysotile asbestos miners*, Occup. Environ. Med. 66:805-809 (2009).
- Turci, et al., *Role of associated mineral fibers in chrysotile asbestos health effects: The case of Balangerolite*, Ann. Occup. Hyg. 53:491-497 (2009).
- Madkour, et al., *Environmental exposure to asbestos-response relationship with mesothelioma*, Eastern Mediterranean Health J., 15:25-38 (2009).
- Yano, et al., *Mesothelioma in a worker who spun chrysotile asbestos at home during childhood*, Am. J. Ind. Med., 52:282-287 (2009).
- Baumann, et al., *Pleural Mesothelioma in New Caledonia: An acute environmental concern*, Cancer Detect Prev., 31:70-76 (2007).
- Baumann, et al., *Pleural Mesothelioma in New Caledonia: Associations with environmental risk factors*, Environ. Health Perspect. 119:695-700 (2011).
- Finkelstein, et al., *Mesothelioma among employees of a Connecticut factory that manufactured friction materials using chrysotile asbestos*, Ann. Occup. Hyg. 54:692-696 (2010).
- Egilman, et al, *A case of occupational peritoneal mesothelioma from exposure to tremolite-free chrysotile in Quebec, Canada: A black swan case*, Am J. Ind. Med. 54:153-156(2011).
- Kanarek, *Mesothelioma from Chrysotile Asbestos: Update*, AEP Vol. 21, No. 9 pp. 688-97(2011).

- Wang, et al., *Cause-Specific Mortality in a Chinese Chrysotile Textile Worker Cohort, J*. Japanese Cancer Assn. (2012).

- Stayner, et al., *The Worldwide Pandemic of Asbestos-Related Diseases,* Annual Reve. Public Health,34:4.1-4.12(2013).

- Roelofs, C, et al, *Mesothelioma and employment in Massachusetts: Analysis of cancer registry data 1988-2003*. American J. Industrial Med. 56:985-992.

- Li, et al. *Cohort studies on cancer mortality among workers exposed only to chrysotile asbestos: a meta-analysis.* Biomedical and Environ Sci. 17:459-468. (2004).

Among various health agencies and scientists, there is general agreement that all forms of asbestos, including chrysotile, cause all asbestos-related diseases, including mesothelioma. This general agreement includes agencies and organizations such as:

- U.S. Public Health Service, U.S. Department of Health & Human Services*. Toxicological Profile for Asbestos, Atlanta*: Agency for Toxic Substance and Disease Registry (ATSDR), September 2001. "exposure to any asbestos type (i.e. serpentine or amphibole) can increase the likelihood of lung cancer, mesothelioma, and nonmalignant lung and pleural disorder."

- American Conference of Governmental Industrial Hygienists. Asbestos: TLV® Chemical Substances 7th Edition Cincinnati, OH: ACG1H; Report No.: Publication #7DOC-040 (2001).

- American Thoracic Society, *Diagnosis and initial management of nonmalignant diseases related to asbestos.* Am. J. Respir. Crit. Care Med.; 170(6):691-715 (Sep. 15,2004).

- Environmental Protection Agency. Airborne Asbestos Health Assessment Update. Springfield VA: NT1S; Report No.: EPA/600/8-84/003F (June 1986).

- National Toxicology Program. Report on Carcinogens, Eleventh Edition. U.S. Department of Health and Human Services, Public Health Service (2004).

- Occupational Safety and Health Administration. Occupational exposure to asbestos; final rule. Federal Register; 59:40964-t 162 (1994).

- Consumer Product Safety Commission. CANCER HAZARD! CPSC Warns About Asbestos in Consumer Products: Safety Alert. Report No.: CPSC Document #5080 (1994).

- World Health Organization. Environmental Health Criteria 203: Chrysotile Asbestos. Geneva: World Health Organization; (1998); World Health Organization. Elimination of asbestos related diseases. Ref Type: Generic (2006); World Health Organization. Environmental Health Criteria S3: Asbestos and Other Natural Mineral Fibres. Geneva: World Health Organization; (1986).

- Collegium Ramazzini, *The Case for a Global Ban on Asbestos*. Environ. Health Perspectives. 118:897-901 (2010).

- World Trade Organization. European Communities - Measures Affecting Asbestos and Asbestos-Containing Products. Report No.: WT/DS135/R (2000).

- IARC: Monograph on the Evaluation of Carcinogenic Risks to Humans 2012; 100C:219-309)

5. DETERMINING THE CAUSE OF MESOTHELIOMA DOES NOT REQUIRE QUANTIFICATION OF ASBESTOS EXPOSURE

Medical professionals determine causation of mesothelioma based upon the qualitative history of asbestos exposure, not by quantitative analysis of that exposure. It has been generally accepted by the medical and scientific community for decades that a history of asbestos exposure is the most reliable evidence upon which to base a causation determination. That is, doctors rely upon the patient's or family members' description of his history of exposure to asbestos. Neither medicine nor science has ever required a calculation of some quantitative dose of asbestos exposure to link a patient's mesothelioma with asbestos exposure.

Reliance on a history of asbestos exposure to establish causation of mesothelioma goes back to the landmark studies of Wagner, Selikoff and Newhouse in the 1960s, all of which attributed mesothelioma to a history of asbestos exposure without any quantitative analysis. To this day, using exposure history to diagnose mesothelioma is an accepted methodology among such health organizations such as the American Thoracic Society, the American Cancer Society, and the National Cancer Institute.

The attribution of a disease to a given cause (Medical Causation), particularly in dealing with a signature disease of asbestos exposure like mesothelioma, is based on the weight of the evidence approach and not on the ability to quantify a given exposure or set of exposures. It is generally accepted in the medical and scientific community that once you have a medical patient diagnosed with mesothelioma with a history of occupational, domestic or para-occupational asbestos exposure, the mesothelioma is attributed to asbestos exposure. Under the Helsinki Criteria (*Consensus Report, Asbestos, Asbestosis and Cancer: The Helsinki Criteria for Diagnosis and Attribution*. Scandinavian Journal of Work and Environmental Health 1997, 23:311-6), assessing disease causation does not require a quantification of an occupational, domestic or para-occupational asbestos exposure because:

a) asbestos exposures at "occupational levels" result in asbestos fiber levels thousands and tens of thousands of times higher than background/ambient air levels;
b) a "safe" or threshold level of exposure to asbestos has never been identified for the disease mesothelioma (discussed below); and
c) asbestos exposures as brief as a few days have been shown to cause mesothelioma.

See Morris Greenberg & T.A. Lloyd Davies, *Mesothelioma Register 1967-1968*, Brit. J. Ind. Med. 91, 91-104 (1974) and Skammeritz E., Olmand LH, Johansen JP, Omland O, *Asbestos Exposure and Survival in Malignant Mesothelioma: A Description of 122 Consecutive Cases at an Occupational Clinic*, 2(4) J. Occupational & Envtl. Med. 228, 228-29 (Oct 2011)

6. A THRESHOLD LEVEL OF ASBESTOS EXPOSURE FOR THE DEVELOPMENT OF MESOTHELIOMA HAS NOT BEEN DEMONSTRATED

Every United States government agency (including OSHA, NIOSH, CDC, NIH and EPA) and every world agency (including IARC, WHO and ILO) that has reviewed the scientific literature concerning asbestos exposures and mesothelioma have concluded that there is no safe level (or threshold) of exposure to asbestos that has been shown not to cause mesothelioma:

- EPA, 1973: "Finally, the available evidence suggests a gradient of effects from direct occupational, to indirect occupational exposure, to indirect occupational exposure to families of workers exposed to asbestos ... [t]his suggests that there are levels of asbestos exposure that will not be associated with any detectable risk, although these levels are not known."

- NIOSH, 1976: "Excessive cancer risks have been demonstrated at all fiber concentrations studied to date. Evaluation of all available human data provides no evidence for a threshold or a “safe” level of asbestos exposure."

- NIOSH, 1980: "All levels of asbestos exposure studied to date have demonstrated asbestos related disease ... there is no level of exposure below which clinical effects do not occur."

- USPHS, 1980: "It is important to point out that when a permissible level for exposure (PEL) to a certain carcinogen is set by OSHA, there is no implication that such a level is safe. To the contrary, it is the agency's policy that any occupational exposure to a carcinogen carries with it some risk of disease, even if it cannot be easily or precisely measured."

- OSHA, 1994: "reducing exposure to 0.1 f/cc would further reduce, but not eliminate, significant risk. The 0.1 f/cc level leaves a remaining significant risk."

- WHO, 1998: "Exposure to chrysotile asbestos poses increased risks for asbestosis, lung cancer and mesothelioma in a dose-dependent manner. No threshold has been identified for carcinogenic risks."

- WHO, 2000: "Asbestos is a proven human carcinogen (IARC Group 1). No safe level can be proposed for asbestos because a threshold is not known to exist. Exposure therefore should be kept as low as possible."

7. BRIEF AND LOW-LEVEL ASBESTOS EXPOSURES CAUSE MESOTHELIOMA

For decades, mesothelioma has been seen in individuals with non-occupational asbestos exposures, dating back to the 1960s. (Wagner 1960, Newhouse and Thompson 1965) The exposures in these articles were sustained by family members of workers who inadvertently brought asbestos dust home on their clothing, and by those who lived in neighborhoods with industrial sources of asbestos, including asbestos mines and plants. In these and other landmark articles, mesothelioma was identified among people who had sustained low and high levels of exposures to asbestos as well as short and long terms of exposures to asbestos. Such findings continue in scientific studies to this day.

The reality of causation with respect to mesothelioma is generally accepted in the scientific community and was summarized in the peer-reviewed article *Asbestos Exposure Causes Mesothelioma, But Not this Asbestos Exposure: An Amicus Brief to the Michigan Supreme Court*, International Journal of Occupational and Environmental Health 2007; 13:318-327, which was authored by Dr. Welch, with 51 other pre-eminent experts in asbestos and asbestos-related disease signing on:

Accepted Method for Evaluating Disease Causation in an Individual: Generally, and as Applied to Asbestos Exposure and Mesothelioma.

Examining the question of causation of disease in an individual generally involves four questions: 1) was the individual exposed to a toxic agent; 2) does the agent cause the disease present in the individual; 3)

was the individual exposed to this substance at a level where disease has occurred in other settings; and 4) have other competing explanations for the disease been excluded?

There is no reasonable dispute regarding Question 2 – asbestos causes mesothelioma. Additionally, there are no well-accepted competing explanations regarding mesothelioma that must be excluded, resolving Question 4. Thus, when considering the issue of causation of a mesothelioma, once an occupational or para-occupational exposure to asbestos has been established (Question 1), the sole question remaining for examination is whether the exposure or set of exposures of that individual is comparable to exposures that have been documented to cause mesothelioma in others – Question 3.

The mainstream scientific community is in consensus regarding the resolution of Question 3. As discussed above, there is no safe level of exposure to asbestos. Even exposure at current regulatory levels results in excess mesothelioma. Accordingly, the consensus of the scientific community is that any occupational or para-occupational exposure to asbestos — even "brief or low-level exposures" – must be considered a medical and scientific cause of a mesothelioma.

8. DOMESTIC ASBESTOS EXPOSURES, I.E. FAMILY MEMBERS EXPOSURES, TO ASBESTOS CAN CAUSE MESOTHELIOMA

Domestic exposures, i.e., a family members' exposures to asbestos from work clothes, shoes, or hair introduced into the home of a worker who worked with or around asbestos, and their resultant disease manifestations are comprehensively outlined in the National Institute for Occupational Safety and Health ("NIOSH") Report to Congress on Worker's Home Contamination Study, which was conducted under The Workers' Family Protection Act (29 U.S.C. 671a). In this report, NIOSH concluded that:

"... families of asbestos-exposed workers have been at increased risk of pleural, pericardial, or peritoneal mesothelioma, lung cancer, cancer of the gastrointestinal tract, and non-malignant pleural and parenchymal abnormalities as well as asbestosis."

As stated above, the scientific and medical community has yet to determine a level of exposure to asbestos below which mesothelioma will not occur. Very low levels of exposure above background have been shown medically and scientifically to cause mesothelioma. It has been repeatedly and consistently demonstrated in the medical and scientific literature that family members exposed to asbestos dust from laundering a worker's clothing have a significantly increased risk of developing mesothelioma. Researchers have confirmed that this risk is substantially, in excess of two times that of the general population. The following are examples of the many studies, texts and reports that support or form the basis for my opinions:

- Wagner, *Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province,* Brit. J. Industr. Med., 17:260-269 (1960).

- Newhouse & Thompson, *Mesothelioma of Pleura and Peritoneum following Exposure to Asbestos in the London Area*, Brit J. Industr. Med. 22:261-269 (1965).

- Leiben & Pistawka, *Mesothelioma and Asbestos Exposure*, Arch. Environ. Health, 14:559-566 (1967).

- Champion, *Two Cases of Malignant Mesothelioma after Exposure to Asbestos*, Am. Rev. Res. Dis. 103(6):821 -826 (1971).

- Lillington, *Conjugal Malignant Mesothelioma [letter]*, New Engl. J. Med., 291(11):581-585 (1974).
- Greenberg & Davis, *Mesothelioma Register 1967-1968*, Brit. J. Med. 31:91-104 (1974).
- Anderson, *Household-Contact Asbestos Neoplastic Risk*, NY Acad. Sci. 271:311-323 (1976).
- Li, Familial *Mesothelioma After Intense Asbestos Exposure at Home*, JAMA 240(5):467 (1978).
- Vianna and Poland, *Non-Occupational Exposure to Asbestos and Malignant Mesothelioma in Women*, Lancet 8073:1061-1063 (1978).
- Epler, *Asbestos-Related Disease from Household Exposure*, Respiration, 39:229-240 (1980).
- Tagnon, *Mesothelioma Associated with the Shipbuilding Industry in Coastal Virginia*, Cancer Research, 40:3875-3879 (1980).
- Hammar, *Familial Mesothelioma: A Report of Two Families, Human Pathology*, 20:1-7-112 (1989).
- Schneider, *Pleural Malignant Mesothelioma and Household Exposure*, Review Environ. Health, 11:65-70 (1996).
- Hillerdal, *Mesothelioma: Cases Associated with Non-Occupational and Low Dose Exposures*, Occup. Environ. Med., 56-505-513 (1999).
- Dodson, *Quantitative Analysis of Asbestos Burden in Women with Mesothelioma*, Am. J. Ind. Med. 43:188-195 (2003).
- Bourdes, *Environmental Exposure to Asbestos and Risk of Pleural Mesothelioma: Review and Mela-Analysis*, European J. of Epi., 16:411-417 (2000) (relative risk of pleural mesothelioma for household exposures ranged between 4.0 and 23.7 and the summary risk estimate was 8.1 with a 95% CI of 5.3 1012).
- Magnani, *Multicentric Study on Malignant Pleural Mesothelioma and Non-Occupational Exposure to Asbestos*, British J. of Cancer, 83(1): 104-111 (2000) (domestic exposure was associated with an increased risk with an Odds Ratio of 4.81 with a 95% CI of 1.8 to 13.1).
- Kradin, *Diffuse Peritoneal Mesothelioma: A Case Series of 62 Patients Including Paraoccupational Exposure to Chrysotile Asbestos*. Am. J. Industrial. Med. 2017. 60:963-967. 50% of paraoccupationally exposed were exposed only to dust from products made with chrysotile asbestos.

9. ASBESTOS EXPOSURES ABOVE BACKGROUND CONTRIBUTE TO THE RISK OF DEVELOPING MESOTHELIOMA

Due to the extensive and longstanding use of asbestos, the ambient air in the United States contains minute amounts of asbestos. Those ambient outdoor air concentrations are generally known as the "ambient" or "background level." Background levels of asbestos have not been epidemiologically proven to cause mesothelioma. While it is theoretically possible that background levels of asbestos could cause mesothelioma, it is impossible to test this, since such proof would require nearly infinitely large comparison groups and it would be impossible to find individuals with less than ambient air exposure.

There is no level of asbestos exposure above background levels that has been shown to not contribute to causing mesothelioma. Occupational or para-occupational (such as household or domestic exposure) exposures necessarily exceed by orders of magnitude the background levels to which individuals are exposed simply by living in the U.S. Thus, occupational and para-occupational exposures, being orders of magnitude above background levels, no matter how brief, contribute to the risk of developing mesothelioma.

An example of a low-level exposure is 0.01 f/cc, which is ten times lower than the current OSHA permissible exposure level (PEL). In contrast, ambient air concentrations or background levels have been reported at 0.0001 to 0.00000001 f/cc, tens of thousands of times less than the current permissible exposure limit, or PEL, of 0.1 f/cc. Thus, even low-level exposures to asbestos exceed background levels by an order of many magnitudes. Put differently, the current OSHA PEL allows 100,000 fibers in a cubic meter of air. The example of a low-level exposure above is 10,000 fibers in a cubic meter of air. The measured ambient levels discussed above reflect levels between 1/100th of a fiber up to 100 fibers in a cubic meter.

Furthermore, OSHA recognizes that even with exposure at the PEL, there will be cases of mesothelioma. (Federal Register 1986, Part II: Department of Labor, Occupational Safety and Health Administration, 29 CFR Parts 1910 and 1926: Occupational exposure to asbestos, tremolite, anthophyllite & actinolite. Final Rules, pages 22612-22790; Table 6 on page 22644) The studies referred to in this report also provide evidence that there can be an increased incidence of mesothelioma at concentrations below the PEL. Therefore, the lowest concentration of asbestos that can produce mesothelioma is currently unknown.

Additionally, epidemiological studies have found that even at the lowest levels of asbestos exposure, there have been increases in the incidence of mesotheliomas. (Iwatsubo, 1998; Rodelsperger, 2001) Furthermore, it is the documented consensus of the international scientific and medical community that an occupational history of brief or low-level exposure is sufficient for a mesothelioma to be designated as occupationally-related. (*Consensus Report, Asbestos, Asbestosis and Cancer: The Helsinki Criteria for Diagnosis and Attribution*. Scandinavian Journal of Work and Environmental Health 1997, 23:311-6)

If a person sustains asbestos exposures above background/ambient levels of exposure as reflected by an occupational, para-occupational and/or domestic asbestos exposure and goes on to develop mesothelioma, it is my opinion that the exposures above background levels, taken in context of the individual's total (cumulative) asbestos exposures, are significant and non-trivial, and are medical and scientific causes in the development of the individual's mesothelioma. It is not my opinion that a "single fiber," or that "each" or "any" exposure to asbestos, even those below background levels, are a substantial contributing factor in causing mesothelioma. To the contrary, as noted above, a single day of exposure at the current OSHA PEL of 0.1 f/cc equates to literally years of exposure to what the ATSDR reports as typical rural ambient asbestos exposures of 0.00001 f/cc.

To expound further on the above paragraph, I am often asked when is an asbestos exposure "significant" or "substantial". While not all inclusive, an asbestos exposure or exposures can be "significant" or "substantial" if:

1) it is of the nature, type and duration that has been shown to cause mesothelioma in the medical and scientific literature; and
2) if it is not trivial in the context of the individual's *total* asbestos exposure, e.g., patching a hole in a wall with joint compound by a 40-year career insulator.

In the legal setting, I am often posed hypotheticals that ask me to assume that an individual was

exposed, for example, to "one fiber" of asbestos or for "one second,"or to asbestos "barely above background." I have never encountered such exposures in practice. It must be emphasized that small quantities of asbestos containing materials may be composed of millions, or billions of asbestos fibers. Support for these numerical values comes from simple mathematical calculations, which are often performed by mineralogy and industrial hygiene experts:

- Arthur Langer, Ph.D., Deposition taken in *Barbara Harris and Dale Harris vs. Bondex International, Inc., et al,* Superior Court of the State of California for the County of Los Angeles, April 18, 2007, pp. 146-150. (Confirming that one gram of chrysotile asbestos contains approximately 80 *billion* asbestos fibers, and one 25 lb. bag of joint compound at 5% asbestos contains *45. 5 trillion* asbestos fibers)

- Eric Chatfield, Ph.D., Deposition taken in *Carl Terranova, et ux., vs. John Crane, Inc. et al., Cause No. 17342-BH01-3*, In the District Court, Brazoria County, Texas, 23rd Judicial District, July 28, 2005. (Confirming his attendance, presentation and associated PowerPoint at the 2005 ASTM Johnson Conference, entitled "Some Measurements of Tremolite Concentrations in Chrysotile from Different Mining Locations" see slide 12 of PowerPoint presentation reflecting mean of over *10 trillion* chrysotile fibers per gram of U1CC-B (Canadian) chrysotile analyzed by Addison/Davies Method and TEM)

Therefore, simple tasks such as cutting (or removing) asbestos-containing pipe covering, mixing asbestos containing dry products, sanding asbestos-containing joint compound, cutting asbestos-containing boards or asbestos-containing pipe, wire-brushing or power-wire brushing adhered asbestos-containing gaskets, or sweeping asbestos containing dust/residue results in exposures to millions, billions and/or trillions of respirable/breathable asbestos fibers.

These numbers can also be put into perspective using a fiber/cc analysis to compare the amount of asbestos fibers inhaled by a worker during a work day with occupational asbestos exposures vs. an individual who is simply breathing in the "normal" "ambient" or "background" air. The following values can be used as a crude comparison of how many asbestos fibers can be inhaled while breathing the ambient air versus working with an asbestos-containing product:

- An individual performing light physical labor, on average, will take 16 breaths/minute.
- An individual at rest or performing light activity will take 12 breaths/minute.
- One breath = 500 cubic centimeters ("cc") of air.

Thus, for an individual exposed at 1.0 fiber/cubic centimeter of asbestos at work:

- 16 breaths x 500 cc of air in one breath x 1.0 fiber/cc = 8,000 fibers/minute;
- At 15 minutes of exposure at 1 fiber per cc: 8,000 x 15 = 120,000 fibers are breathed;
- At 1 hour of exposure at 1 fiber per cc: 8,000 x 60 minutes = 480,000 fibers/hour are breathed;
- One working day of exposure at 1.0 fiber per cc: 480,000 x 8 = 3,840.000 fibers/day are breathed.

At the current OSHA permissible exposure level (PEL) of 0.1 f/cc, an individual would breathe 384,000 fibers in one day. At the low-level exposure of 0.01 f/cc, which is ten times lower than the current OSHA permissible exposure level (discussed supra), an individual would breathe 38,400 fibers in one day.

In comparison, for an individual who is simply breathing in the "normal" "ambient" or "background" air:

- Resting = 12 breaths/minute;
- 12 breaths x 500 cc of air = 6,000 cubic centimeters of air/minute or 360,000 cubic centimeters of air/hour,

- 360,000 cubic centimeters of air/hour x 24 hours = 8,640,000 cubic centimeters of air/day;
- 8,640,000 x .00001 f/cc (ATSDR values) = 86 asbestos fibers in one day

Extrapolating this even further, an individual breathing the "normal", "ambient" or "background" level of .00001 fibers/cc of asbestos over a lifetime of 80 years:

- 86 fibers/day x 365 days x 80 = 2.5 million fibers in a lifetime of 80 years.

Therefore, just one day of occupational exposure at 1.0 fiber/cc of asbestos is greater than a lifetime of "ambient" or "background" levels of asbestos exposure**.** Furthermore, it is important to recognized that ambient exposures occur at such low levels that the host defense systems can limit them in a far different manner than when working with the millions of fibers released during the course of occupationally work. Therefore, a lifetime accumulation of a dose of e.g. 0.1 f/cc-years *should not* be compared to work for a several hours at a concentration of, e.g., 0.1 f/cc. Despite the idea that asbestos-related diseases are dose-dependent, the intensity of the dose is the greatest determinant of whether the body's defense mechanisms will ultimately limit toxic biological effects. None of the calculations presented here are novel; they simply reflect basic pulmonary physiology contrary.

As reflected in the calculations above, nobody is ever exposed to a "single fiber" of asbestos or exposed "just above background" through occupational and para-occupational exposure. Such hypothetical questions have no real-world scientific meaning.

The following are additional examples of the many studies, texts and reports that support or form the basis for my opinions:

- *Champion P. Two Cases of Malignant Mesothelioma After Exposure to Asbestos*, American Review of Respiratory Disease 1971 ;105 ('There is probably no really safe level of asbestos exposure...It is clear that only minor exposure is required for the development of malignant tumors...")

- Greenberg and Davies, *Mesothelioma Register 1967-1968*, British Journal of Industrial Medicine, 1974;91-104 ('in this study the briefest occupational exposure to asbestos associated with a mesothelial tumour *was three weeks, but if asbestos was a cause of mesothelioma it cannot be assumed that lesser exposures are safe."* p. 103)

- Selikoff I. *The Asbestos Exposure of Insulation Workmen, Insulation Hygiene Progress Reports 1975*;6(l): l-4 ("short-term dust concentrations during specific insulation practices can be extremely high..." p. 4)

- NIOSH. Revised Recommended Asbestos Standard 1976, p. 55 (reporting association between mesothelioma and "occupational exposures in some cases as brief as one day")

- Chen and Mottet, *Malignant Mesothelioma with Minimal Asbestos Exposure, Human Pathology* 1978;9(3):253. ("Estimation of the number of asbestos fibers in the lungs suggests the low-level exposure and establishes that, for some individuals at least, slight exposure to asbestos can result in malignant mesothelioma.")

- *NIOSH-OSHA Asbestos Work Group, Workplace Exposure to Asbestos, Review and Recommendations*, DHHS (NIOSH) Pub. No. 81-103,1980 ("Excessive cancer risks, however, have been demonstrated at all fiber concentrations studied to date. Evaluation of all available human data provides no evidence for a threshold or for a "safe" level of asbestos exposure.... (The absence of a threshold is further indicated by

the dramatic evidence of asbestos-related disease in members of asbestos-worker households and in persons living near asbestos-contaminated areas. These household and community contacts involved low level and/or intermittent casual exposure to asbestos. Studies of duration of exposure suggest that even at very short exposure periods (1 day to 3 months) significant disease can occur." p. 3)

- Committee on Nonoccupational Health Risks of Asbestiform Fibers, Board on Toxicology and Environmental Health Hazards, Commission on Life Sciences, National Research Council*, Asbestiform Fibers Nonoccupational Health Risks*, National Academy Press, Washington, D.C. 1984, p.212 (background environmental exposure of 0.0004 f/cc over a 73-year lifetime was associated with 9 cases of mesothelioma per million. A "higher** exposure of 0.002 f/cc was associated with 46 cases of mesothelioma per million - a five-fold risk.)

- Pairon C, Orlowski E, Iwatsubo Y, et al. *Pleural Mesothelioma and Exposure to Asbestos: Evaluation from Work Analysis of Asbestos Bodies in Bronchoalveolar Lavage Fluid or Lung Tissue in 131 Patients*, Occupational and Environmental Medicine 1994; 51:244-249 ("Ilgren and Browne considered whether a threshold exposure might exist and concluded that mesothelioma was unlikely in persons exposed for less than 5 f/ml-years. Our results indicate, however, that mesothelioma cases occurred below a cumulative exposure of 5 f/ml-years and perhaps below 0.5 f/ml-years." p. 141)

- NIOSH. *Report to Congress on Workers' Home Contamination Study Conducted Under the Workers' Family Protection Act 1995* (reviewing twelve epidemiology studies and multiple case reports and concluding, "Mesothelioma has occurred following short term asbestos exposures of only a few weeks and can result from very low levels of exposure." p. 7)

- Frank A. *The Riddle of Risk Assessment in Asbestos Carcinogenicity, Med Lav.* 1997;88(4):333-338 ("There is no safe level of asbestos' -I.J. Selikoff, MD, Third Wave Conference" p. 333) ("there is no safe level of exposure to asbestos, leading to the conclusion that there is no threshold for this carcinogenic substance, just as there does not appear to be a threshold for other carcinogens." p. 335)

- Iwatsubo Y, Pairon JC, Boutin C, et al. *Pleural Mesothelioma: Dose-Response Relation at Low Levels of Asbestos Exposure in a French Population-Based Case-Control Study,* American Journal of Epidemiology 1998; 148(2): 133-148 (Abstract: "...We found a clear dose-response relation between cumulative exposure to asbestos and pleural mesothelioma in a population-based case-control study with retrospective assessment of exposure. A significant excess of mesothelioma was observed for levels of cumulative exposure that were probably far below the limits adopted in many industrial countries during the 1980s.")

- Hillerdal, J. *Mesothelioma: Cases Associated with Non-Occupational and Low Dose Exposures, Occupational and Environmental Medicine* 1999;56:505-513 ("Results and Conclusions—There is no evidence of a threshold level below which there is no risk of mesothelioma. Low level exposure may include peak concentrations which can be very high for short periods. There might exist a background level of mesothelioma occurring in the absence of exposure to asbestos, but there is no proof of this....").

- Hodgson JT, Damton A. *The Quantitative Risks of Mesothelioma and Lung Cancer in Relation to Asbestos Exposure, Annals of Occupational Hygiene* 2000;44(8):565-601. ("The attempt (Ilgren and Browne, 1991) to deduce a 'threshold' by identifying the lowest estimated dose received by any observed case is a logical nonsense. ... [W]e do not believe there is a good case for assuming any threshold for mesothelioma risk." p. 583)

- World Trade Organization, *European Communities - Measures Affecting Asbestos and Asbestos-Containing Products, Report of the Panel*, WT/DS 1135/R/18 September 2000 ("it is scientifically

accepted that there is no biological threshold of harmlessness.... The 1998 WHO report carried out under the International Programme on Chemical Safety states that, for chrysotile: "No threshold has been identified for carcinogenic risks", p. 35)

- Agudo, et al., *Occupation and Risk of Malignant Pleural Mesothelioma: A Case-Control Study in Spain*, Am. J. Ind. Med. 37:159-168 (2000) ("Compared to those who never worked or who were considered as never exposed, all levels of probability and intensity had an increased significant risk, except subjects with low probability of exposure."

- Rodelsperger C, Jockel K, Pohlabeln H, et al. *Asbestos and Man-Made Vitreous Fibers as Risk Factors for Diffuse Malignant Mesothelioma: Results From a German Hospital-Based Case-Control Study*, American Journal of Industrial Medicine 2001; 39:262-275

- Odds ratio of 7.9 CI 2.1-30.0 for cumulative exposures of greater than 0.00 up to 0.15 f/cc-years and odds ratio of 21.9 for cumulative exposures between 0.15 and 1.5 f/cc-years.

- Despite the possible influence of selection and information bias, our results confirm the previously reported observation of a distinct dose-response relationship even at levels of cumulative exposure below 1 fiber year. (p. 262)

- A risk estimate based on accurate workplace measurements is not yet available. Nevertheless, it has recently been demonstrated that an increase of risk may occur even below a cumulative exposure of a few fiber years (fibers/mL x years) [Iwatsubo et al, 1998]. (p. 263)

- British Thoracic Society Standards of Care Committee, *Statement on Malignant Mesothelioma in The United Kingdom*, Thorax 2001; 56:250-265 ("There is no evidence for a threshold dose of asbestos below which there is no risk." p. 252)

- U.S. Public Health Service, U.S. Department of Health & Human Services. *Toxicological Profile for Asbestos*, Atlanta: Agency for Toxic Substance and Disease Registry (ATSDR), September 2001.

- Galateau-Salle F, ed. *Pathology of Malignant Mesothelioma*, Springer Science + Business Media, 2006 ("Substantial numbers of mesotheliomas are now seen as a result of nonoccupational exposures, including occasional "handyman' type exposure, domestic exposure (e.g., from laundering asbestos-contaminated work clothes), and other types of occasional or non-occupational exposures. Mesothelioma has been reported to occur after brief low-level or indirect exposure.)

- Dail and Hammar's Pulmonary Pathology (3rd ed.), 2008, Vol. II Neoplastic Lung Diseases, Ch. 43 ("when there are multiple asbestos exposures, each contributes to cumulative exposure and hence to the risk of causation of MM [malignant mesothelioma], with any appropriate latency interval" p. 587)

- Skammeritz E., Olmand LH, Johansen JP, Omland O, *Asbestos Exposure and Survival in Malignant Mesothelioma: A Description of 122 Consecutive Cases at an Occupational Clinic,* 2(4) J. Occupational & Envtl. Med. 228 (Oct 2011) ("Each patient with known exposure was categorized by an experienced occupational physician, based on the intensity of cumulative exposure, as "low" (<10 fibers/cm^3-year) (p. 226). Of the patients [with mesothelioma] with documented exposure, 51 (47.6%) had low cumulative exposure (pp. 228-229). The total time of exposure ranged from a few days to over 40 years...It has never been possible to establish a lower threshold for cumulative asbestos exposure in relation to the development of Malignant Mesothelioma! (p. 231))

- IARC Monograph on the Evaluation of Carcinogenic Risks to Humans 2012; 100C:219-309

- Offermans, et al., *Occupational Asbestos Exposure and Risk of Pleural Mesothelioma, Lung Cancer, and Laryngeal Cancer in the Prospective Netherlands Cohort Study*, Journal of Occupational and Environmental Medicine, 2014 ("For mesothelioma, [Hazard Ratios] were significantly elevated in this study, even for the lowest tertile of CE (median, 0.20 f-y/mL) based on FINJEM (HR=2.69 [95% CI, 1.60 to 4.53]) (p. 15)

- Lacourt, et al., *Occupational and Non-Occupational Attributable Risk of Asbestos Exposure for Malignant Pleural Mesothelioma*. Thorax, 2014 (Cumulative exposures >0-0.1 f/mL-years showing a 4 times excess risk for pleural mesothelioma (Table 4))

The retrospective estimates in the above articles are not relied upon for a quantitative dose level or precise threshold needed for causation, but rather to illustrate that the scientific and medical communities recognize that a history of brief or low-level asbestos exposure is sufficient to establish causation.

10. GENETICS OF MALIGNANT MESOTHELIOMA

Like other cancers, malignant mesothelioma reflects a series of genetic mutation events that lead to the uncontrolled growth of a clone of malignant cells. It takes on average between 20-40 years for the disease to become clinically evident following exposure to asbestos. Whereas some epidemiologists have suggested that early exposures may be more important in causing disease than later ones (Peto et al and Bofetta et al), the current understanding of the mutational requirements for developing mesothelioma would suggest that all exposures occurring after ten years of first exposure (the minimal latency as suggested by the Helsinki Consensus Conference) can potentially contribute to mutagenesis and must be considered as potentially causative of malignant mesothelioma. In this regard, the modeling of disease by epidemiologists cannot be used to exclude the effects of later exposures. For example, individuals exposed to low levels of asbestos via para-occupational exposures in childhood, and who subsequently worked occupationally with asbestos, cannot be said to have had their mesothelioma caused by early exposures alone. The biology of carcinogenesis excludes that premise.

Many mesotheliomas are polyclonal neoplasms possibly reflecting a “field effect” induced by asbestos. The Cancer Genome Atlas program studied 74 mesotheliomas form genetic alterations using next-generation sequencing (NGS), whole-exome sequencing (WES), messenger RNA expression, methylation analysis, microRNA expression, exomes, reverse-phase protein array, and transcription factor analyses. They observed frequent mutations in *CDKN2A*, *NF2*, *TP53*, *LATS2* and *SETD2*. Mansfield et al suggested that genetic alterations in mesothelioma may yield neoantigens that are immunogenic and may be susceptible to immunotherapeutic approaches.

Malignant mesothelioma are heterogeneous with complex genetic, chromosomal, and epigenetic changes. They display a relatively low mutation burden compared to most adult solid tumors. (Blanquart, Jaurand, & Jean, 2020) Genetic sequencing of tumors, in situ hybridization, and immunohistochemical techniques have demonstrated that greater than 90% of mesothelial malignancies show mutations in the *CDKN2A* gene, and roughly half show characteristic mutations in *BAP-1.* However, there is currently no evidence that neoplastic mutations occur spontaneously in nature in the absence of an additional environmental stimulus, e.g., asbestos.

Currently, there are several genetic mutations that appear to occur with increased frequency in patients with mesothelioma. These include loss of *BAP-1* expression (a member of the BRAF family), and p16

deletions. The loss of *BAP-1* is common as an acquired somatic mutation (~50% of epithelioid mesotheliomas) but can occur as a germ-line (non-acquired) defect in rare families. In a recent comprehensive review of the topic, Cheung and Testa (Cheung and Testa, *BAP-1: A tumor-suppressor gene driving malignant mesothelioma*. 2017.Translational Lung Cancer Research. 6: 270–278) noted that two U.S. families showed a high incidence of malignant mesothelioma, melanoma, and other types of cancers, such as renal carcinoma. Germline *BAP-1* mutations were examined in malignant mesothelioma patients with a family history of cancer, 50 asbestos-exposed control individuals with a family history of cancers other than mesothelioma, and 153 asbestos-exposed control individuals without familial cancer. No *BAP-1* mutations were identified in the control cohorts, but they were present in 9 of 150 (6%) individuals with a family history of cancer. The median age at mesothelioma diagnosis was significantly younger among the 9 *BAP-1* mutation carriers as compared to non-carriers (58 *vs* 68 years) similar to that observed in other cancer predisposition syndromes. There was an overrepresentation of patients with DPM (5 of 9) These individuals appear to have a better overall survival after MM diagnosis (60 *vs.*17 months among the non-carriers), although other studies do not support this claim.

Studies of mice show a high incidence of malignant mesothelioma in asbestos injected *BAP-1* mutant mice, suggesting a very strong role for gene-environment interaction (i.e., exposure to asbestos). *BAP-1* knockout (KO) mice to asbestos-induced malignant mesothelioma resulted from exposures to relatively low doses of asbestos, while there was no compelling evidence that *BAP*-1 KO mice developed mesothelioma in the absence of asbestos exposure. Yoshikawa et al found that chromothripsis, i.e., chromosome shattering yielding random chromosomal rearrangement may be induced by environmental stimuli, e.g. asbestos, and account for the multiple genetic alterations observed in mesothelioma.

For these reasons, it appears that patients with *BAP-1* germline mutations are predisposed to developing mesothelioma following exposure to asbestos, and that spontaneous mesotheliomas in the absence of environmental exposures may not develop.

Other genetic abnormalities have been noted in patients with malignant mesothelioma. Studies by Bueno et al. using next generation sequencing (NGS) and by Hmeljak et al. by Whole Exome Sequencing (WES) have shown mutations in *CDKN2A* (p16), *NF2, TP53, LATS2* and *SETD2,* and it has been suggested that the development of mesothelioma could be limited to mutations in this limited number of genes. (Carbone et al. Mesothelioma scientific clues for prevention, diagnosis, and therapy. Ca Cancer Clin. 2019. 69:402-429) Shih and Kradin have noted patients with mesothelioma who have genetic changes (*MLH1* and *MLH3* deletions) associated with Lynch Syndrome. (*Malignant mesothelioma in Lynch syndrome: A report of two cases and a review of the literature*. Am. J. Indust. Med 62(5):448-452.)

Badhai et al demonstrated that concomitant deletion of the *BAP-1, NF2,* and *CDKN2A* genes in mice results in the development of mesothelioma in all animals, and the knock-out of *CDKN2a* and *NF2* yields mesothelioma in 75%. (Badhai et al. *Combined deletions of BAP-1, NF2, and CDKN2A causes rapid onset of malignant mesothelioma in mice*. 2020. Vol 217 JEM) Knock-out of each these genes individually did not produce mesotheliomas. This suggests that mutations in at least two genes are required for the development of mesothelioma. The authors did not examine whether additional mutations were induced in the mesotheliomas of the affected mice. Although the authors suggest that they can produce mesothelioma in the absence of asbestos, there is no evidence that these mutations occur together spontaneously in nature, nor do the authors consider the likelihood that exposure to asbestos can cause these mutations.

Recent studies suggest that the application of immunostains for MTAP (metalloadenosylphosphorylase) can serve as a reliable marker for expression of *CDKN2A/p16*. Loss of MTAP expression by immunostaining strongly suggests malignant transformation. In a similar vein, loss of *BAP-1* expression by immunostaining correlates well with loss of *BAP-1* gene and is a separate marker of neoplastic

transformation. However, loss of *BAP-1* by immunostaining does not distinguish between somatic and germline loss of *BAP-1*. The concomitant loss of *MTAP* and *BAP-1* by immunostaining is both sensitive and specific for the diagnosis of malignant mesothelioma in tissue and cytology specimens. (Klebe et al)

11. PERICARDIAL MESOTHELIOMA

Pericardial mesothelioma (PM) is a rare malignancy that accounts for 0.7% of all malignant mesotheliomas. The tumor characteristically presents as circumferential growth around the underlying heart and must be distinguished from malignant mesothelioma arising in the mediastinal pleura with extension into the pericardium. The disease has a poor prognosis, with 22% alive at one year and 9% at five years. There has been controversy as whether asbestos is a cause of pericardial mesothelioma, as a history of exposure has been inconsistent. To my knowledge, exposures to cosmetic talc were not determined in the reported cases.

However, Mensi et al identified 8 cases (6 men and 2 women), with a median age at diagnosis of 55.5 years, representing 0.3% of all mesothelioma cases (n = 3,059). The age-standardized incidence rate was 0.09 per million/year. Occupational exposure to asbestos was documented in 5 of the 7 cases for which we obtained an interview. They interpreted their findings to support the role of asbestos in the pathogenesis of PM. (*Pericardial mesothelioma and asbestos exposure.* Int J Hyg Environ Health. 2011. 214:276)

12. COSMETIC TALC PRODUCTS

Talc is a magnesium silicate. Both industrial and cosmetic talcum powders can be contaminated with amphibole and serpentine asbestiform minerals. Whereas industrial talc varies in composition and may include a mixture of mineral particles, cosmetic talc consists of pure platyform particles. In 1974, Rohl and Langer showed that 10 of 20 talc products, including baby powders, facial talcums, and a pharmaceutical, contained both tremolite and anthophyllite asbestiform fibers. (Rohl AN, Langer A. *Identification and Quantitation of Asbestos in Talc*. Enviro Health Persp 1974:9; 5-109)

Blount examined the amphibole content of high-grade cosmetic talc products from deposits in Vermont, Montana, North Carolina and Alabama using a centrifuge/optical method and found a uniformly low amphibole count that included both cleavage fragments and asbestiform fibers with aspect ratios >3:1. (Blount, *Amphibole content of cosmetic and pharmaceutical talcs.* 1991. Environmental Health Perspectives. 94: 225-230)

An internal memo entitled *Cyprus ore reserves-arsenic &* tremolite (03-25-1992) raises concern for the presence of asbestos contamination of the Cyprus West Windsor reserves which was a source of *Johnson & Johnson* (*J&J*) cosmetic talc.

A *J&J* internal memo in April, 1973, noted that "occasionally, sub-trace quantities" of minerals had been found in *Johnson's Baby Powder* that "might be classified as asbestos fiber." *J&J* ran experiments aimed at finding ways to remove asbestos from talc ores. I Vernon Zeitz, head of research and development for Windsor Mineral's *J&J*'s talc mining, said that eliminating asbestos contamination "is strongly urged by this writer to provide the protection against what are currently considered to be materials presenting a severe health hazard and are potentially present in all talc ores in use at this time."

Gordon *et al* tested cosmetic talcum powder and the lung tissue of a woman with malignant mesothelioma and anthophyllite and tremolite by electron dispersive spectrometry (EDS) and selected-area electron diffraction (SAED). (Gordon RE, Fitzgerald S, Millette J. *Asbestos in commercial cosmetic talcum powder as a cause of mesothelioma in women.* Int J Occup Environ Health. 2014 Oct;20(4):318-32)

Dr. William Longo has currently tested, or directed the testing, of 118 containers of *Johnson & Johnson* cosmetic talc for the presence of asbestos. These samples were supplied from the historical inventory of *J&J* from dates between the 1960s and the 1990s and were sourced from Italian (1960s) or Vermont mines (1970s-1990s). Samples were analyzed by polarized light microscopy (PLM), analytical transmission electron microscopy (ATEM) and X-ray diffraction (XRD). To date 51/72 (71%) containers of *J&J* talc samples manufactured in the U.S. prior to 2003, 11/24 (46%) containers manufactured after 2003, for a combined total of 62/96 (65%), were demonstrated to contain amphibole asbestos using TEM with heavy liquid separation. (MAS, May 2020) Using a distinct method developed by a *J&J* consultant at the Colorado School of Medicine, Longo et al have also identified chrysotile asbestos in 6 containers of *Johnson's Baby Powder*. In personal air-sample studies designed to recreate the conditions of "below the waist" applications of *Johnson's Baby Powder*, a mean tremolite concentration of 2.57 f/cc was measured in the ambient air of the subjects breathing zone. (MAS, September 2017)

In a separate study by Longo et al at MAS, all fifteen samples of *Jean Nate* cosmetic talc were demonstrated to show contamination with amphiboles and/or chrysotile asbestos.

Longo has studied *Avon* cosmetic talc (2018) and has demonstrated that >90% of samples are contaminated with amphibole asbestos.

MAS has examined 91 bottles of *Cashmere Bouquet* cosmetic talc (2018) and shown that >90% are contaminated with cosmetic talc.

MAS testing of a variety of *Chanel* talcum products (43) sourced from Italy, Australia, and China showed asbestos (tremolite and/or chrysotile) contamination in >92% of talcum products.

MAS has also tested 21 *Gold Bond* talcum products sourced from Barret's Minerals Montana or Imerys Montana mines. All showed contamination with chrysotile.

The WHO IARC Working Group 100C Monograph Asbestos (Chrysotile, Amosite, Crocidolite, Tremolite, Actinolite, and Anthophyllite) concluded that there is a causal association between exposure to asbestos and cancer of the ovary, based on five strongly positive cohort mortality studies of women with heavy occupational exposure to asbestos. (Acheson et al., 1982; Wignall & Fox, 1982; Germani et al., 1999; Berry et al., 2000; Magnani et al., 2008). Their conclusion received additional support from studies showing that women and girls with environmental, but not occupational exposure to asbestos (Ferrante et al., 2007; Reid et al., 2008, 2009) had positive increases in both ovarian cancer incidence and mortality.

A recent study of 250,000 cosmetic talc users showed no significant increase in risk of developing ovarian cancer after cosmetic talc use, but its findings do not negate the link between asbestos exposure and ovarian cancer. Whereas it does suggest that the *prospective* risk of developing ovarian cancer following exposure to cosmetic talc is low, it suggests that the risk appears to increase with age and when there is a patent gynecological tract. By the authors' admission, their study may not have been optimally powered to detect a statistically increased risk of developing ovarian cancer. (O'Brien KM, Tworoger SS, Harris HR, et al. *Association of powder in the genital area with risk of ovarian cancer. JAMA.* 2020.323).

In February of 2020, Steffen et al reported 10 cases of serous carcinoma of the ovary in subjects exposed to *Johnson & Johnson* cosmetic talc. (J. Occup. Environ. Med.). They found that 8/10 containers were contaminated by tremolite and/or anthophyllite. The investigators supplied detailed dose reconstructions in their study (0.36-5.18 fiber years) and detected anthophyllite and tremolite in tissue analyses (40% and 60% of cases, respectively).

In 2019, Moline et al reported on 33 patients with malignant mesothelioma who were exposed to cosmetic talc products (*Mesothelioma Associated with the Use of Cosmetic Talc.* J. Occup Med. 2019 Oct 10).

In October of 2019, *J&J* voluntarily recalled a lot of *Johnson's Baby Powder* after FDA Laboratory testing showed contamination by asbestos (chrysotile).

In March of 2020, Emory, Maddox and Kradin (Amer J Indust Med) reported 75 subjects with malignant mesothelioma of the pleura, peritoneum, and heart, whose only known exposures to asbestos were through repeated use of cosmetic talcum powders. 11 of the subjects showed evidence of tremolite and/or anthophyllite in non-tumorous tissues.

The following represent a sampling of publications related to this topic:

- Cralley LJ, Key MM, Groth DH, Lainhart WS, Ligo RM. *Fibrous and Mineral Content of Cosmetic Talcum Products*. Am Indus Hyg Assoc Journal 1968 July/August pp 350 -354.
- Rohl AN. *Asbestos in Talc*. Enviro Health Persp 1974 Vol 9 pp 129-132.
- Rohl AN, Langer A. *Identification and Quantitation of Asbestos in Talc*. Enviro Health Persp 1974:9;5-109.
- *Asbestos Found in Ten Powders*. New York Times March 10, 1976.
- Rohl AN, Langer AM, Selikoff IJ, Tordini A, Klimentidis R. *Consumer Talcums and Powders: Mineral and Chemical Characterization.* Journal of Tox and Enviro Health 1976 2:255-284.
- Gordon RE, Fitzgerald S, Millette J. *Asbestos in commercial cosmetic talcum powder as a cause of mesothelioma in women.* Int J Occup Environ Health. 2014 Oct;20(4):318-32.
- Gordon, RE. Cosmetic Talcum Powder as a Causative Factor in the Development of Diseases of the Pleura.*TechOpen.*
- Roggli, V. et al. *Talc and mesothelioma: mineral fiber analysis of 65 cases with clinicopathological correlation.* 2020. Ultrastructural pathology. Vol. 44
- Paoletti L, Caiazza S, Donelli G, Pocchiari F. *Evaluation by Electron Microscopy Techniques of Asbestos Contamination in Industrial, Cosmetic and Pharmaceutical Talcs* Regulatory Toxicology and Pharm 1984 4 222-235.
- Andrion A, Bosia S, Paoletti L, Feyles E, Lanfranco C, Bellis D, Mollo F. *Malignant Peritoneal Mesothelioma in a 17-year-old boy with evidence of previous exposure to Chrysotile and Tremolite Asbestos*. Hum Pathol. 1994 Jun;25(6):617-22.

- Srebo S, Roggli V. *Asbestos-Related Disease Associated with Exposure to Asbestiform Tremolite*. Am J Int Med 1994 26:809-819.

- Steffen J et al. Serous ovarian cancer caused by exposure to asbestos and fibrous talc in cosmetic talc powders. JOEM. 2020. Vol 2. E65-77.

13. CASE-SPECIFIC CAUSATION OPINIONS:

ANTHONY H. VALADEZ DOB:09-23-1998

Materials Reviewed: Received November 12, 2022: A cover letter with a three-paragraph exposure history, pathology reports from Mercy Medical Center and Stanford Medical Center, the deposition testimony of A. Valadez (four volumes), and 54 stained slides, labeled SHS22-7213 (8), SHC22-1269 (1), SHC22-1203 (1), and 22MS-133 (24).

Medical History: The medical history is gleaned from the pathology report from Stanford University Medical Center.

In 2020, Mr. Valadez was a 21-year-old man who presented to physicians with cough and dyspnea and was found to have a pericardial effusion.

In January 2022, he presented with cervical lymphadenopathy and chest CT showed pericardial effusion and tumor masses.

Lymph node biopsy showed malignant mesothelioma.

According to his deposition testimony, he has received both chemotherapy and immunotherapy.

Unfortunately, his prognosis is poor.

Exposure History: Mr. Valadez was repeatedly exposed to *Johnson's Baby Powder* beginning as an infant and continued to apply the powder multiple times daily from the time that he was a teenager, and he also added the powder occasionally to his shoes.

No additional exposures to asbestos have been documented.

Pathology: My review of both the diagnostic pathology shows biphasic malignant mesothelioma in the mediastinum and metastatic to multiple lymph nodes.

Immunostains show that the tumor cells are immunopositive for cytokeratins AE1/AE3, calretinin, CK5/6, WT-1, CK7, and D2-40, and immunonegative for TTF-1, MOC-31, PAX-8, and BerEp4. Nuclear immunostaining of BAP-1 is retained, excluding a possible germline mutation.

No lung tissue was sampled.

Conclusion: Mr. Valadez is a young man who has been diagnosed with diffuse mesothelioma that appears to be primary to the pericardium. However, the presence of tumor in the mediastinum raises concerns that the primary site may be in the mediastinal pleura with secondary extension into the pericardium. An expert radiologist should be asked to review the radiographs with this question in mind.

Despite controversy as to the causative role of asbestos in cases of pericardial mesothelioma, in the presence of the documented history of exposure to cosmetic talc in this case, it is my opinion to a reasonable degree of medical probability that asbestos played a causative role in Mr. Valadez's mesothelioma.

I would also request the opportunity to review the medical records and radiographic reports as well as any reports by expert radiologists tasked with discerning the primary site of Mr. Valadez's tumor prior to giving sworn testimony.

I maintain the right to modify the opinions in this report should additional sources of exposure to asbestos become known prior to my giving sworn testimony.

If I can be of further assistance, please let me know.

Sincerely yours,

Richard Kradin, M.D. (signed electronically)

Richard Kradin, M.D.

RK:sc

Exhibit 2

Anthony Michael Hernandez-Valdez

Patient Health Summary, generated on Dec. 13, 2022

Patient Demographics - Male; born Sep. 23, 1998

Patient Address	Communication	Language	Race / Ethnicity	Marital Status
2695 Agnes Way *(Home)* Merced, CA 95340-3133 *Former (Jan. 18, 2022 - Jan. 17, 2022):* 2695 Agness Way *(Home)* Merced, CA 95340 *Former (Mar. 07, 2017 - Jan. 17, 2022):* 2695 Agnes Way *(Home)* MERCED, CA 95340	[redacted] (Mobile) [redacted] (Home) [redacted] (Work) [redacted]	English (Preferred)	Other Race / Hispanic or Latino	Single

Note from Stanford Health Care and University Healthcare Alliance

This document contains information that was shared with Anthony Michael Hernandez-Valdez. It may not contain the entire record from Stanford Health Care and University Healthcare Alliance.

Allergies

Prochlorperazine (Other (Specify with Comments))

Fosaprepitant (Shortness of Breath)

Oxycodone (Itching, pruritus)

Ondansetron Hcl (Shortness of Breath, Nausea, Vomiting, Dizziness, Headache)

EXHIBIT 2

Component	Value	Ref Range	Abnormality	Analysis Time	Performing Organization	Authorizing Provider
AST (SGOT), Ser/Plas	31	10 - 50 U/L		10/07/2022 12:58 PM PDT	SHC LAB - HOSPITAL LABORATORY	
ALT (SGPT), Ser/Plas	20	10 - 50 U/L		10/07/2022 12:58 PM PDT	SHC LAB - HOSPITAL LABORATORY	
Globulin	4.2	2.0 - 5.0 g/dL		10/07/2022 12:58 PM PDT	SHC LAB - HOSPITAL LABORATORY	

Specimen (Source)	Anatomical Location / Laterality	Collection Method / Volume	Collection Time	Received Time
Blood	BLOOD SPECIMEN / Unknown	Butterfly / Unknown	10/07/2022 11:42 AM PDT	10/07/2022 12:19 PM PDT

Narrative

SHC LAB - HOSPITAL LABORATORY - 10/07/2022 12:58 PM PDT
Physiological plasma concentrations of Sulfasalazine and/or Sulfapyridine drugs may lead to false results for AST and ALT. Please contact the Chemistry section of the clinical laboratory for any questions.

Authorizing Provider	Result Type
Mohana Roy	LAB BLOOD ORDERABLES

Performing Organization	Address	City/State/ZIP Code	Phone Number
SHC LAB - HOSPITAL LABORATORY	300 Pasteur Drive	STANFORD, CA 94305	877-717-3733

CT CHEST ABDOMEN PELVIS W IV CONTRAST - Final result (10/07/2022 11:01 AM PDT)

Anatomical Region	Laterality	Modality
Chest Abdomen Pelvis		Computed Tomography

Specimen (Source)	Anatomical Location / Laterality	Collection Method / Volume	Collection Time	Received Time
			10/07/2022 12:10 PM PDT	

Impressions

10/07/2022 2:54 PM PDT
IMPRESSION:

1. New and enlarging pleural and pericardial nodules.
2. Heterogenous enhancement of the liver, likely representing congestive hepatopathy, which limits evaluation. However, there appears to be a new right hepatic mass measuring up to 3.9 cm. Consider liver MRI for further evaluation if clinically warranted.
3. Stable lymphadenopathy above the diaphragm.
4. Persistent but decreased left internal jugular and innominate vein thrombus.
5. Bilateral pleural effusions with peripheral pleural enhancement, which may represent empyema or malignant effusion.
6. Improved interlobar septal thickening, edema, and opacities.

Michael Nguyentat MD discussed these results with Dr. Roy MD by phone on the following date and time: 10/7/2022 14:05

I have personally reviewed the images for this examination and agree
with the report transcribed above.

Signed"Final report"

Narrative

10/07/2022 2:54 PM PDT

Narrative

CT CH[illegible]

CLINICAL HISTORY: 24 years of age, Male, pericardial epithelioid mesothelioma, status post pericardiectomy and Pleurx placement on 2/17/2022, here for follow-up -he is status post 2 cycles of chemotherapy and 3 cycles of immunotherapy with nivolumab and ipilimumab.

COMPARISON: 8/8/2022

PROCEDURE COMMENTS: CT of the chest, abdomen, and pelvis was performed following administration of iopamidol (ISOVUE 370) 76 % injection 130 mL : 130 mL IV contrast. Oral contrast was not administered prior to the examination.

Dose information: Based on a 32 cm phantom, the estimated radiation dose (CTDIvol [mGy]) for each series in this exam is 9.7 and 5.4. The estimated cumulative dose (DLP [mGy-cm]) is 87.

FINDINGS:

Cardiovascular: Enlarging pericardial nodules, for instance, anterior pericardial nodule at the level of the main pulmonary vein (3/43) measures 2.4 x 1.3 cm, previously 1.7 x 1.1 cm. Some of these nodules demonstrate central necrosis, for instance left anterior pericardial nodule (3/49). Small pericardial effusion. Persistent but decreased thrombus in the left internal jugular and left innominate veins (3/28).

Lung parenchyma, pleura, and airways: Enlarging pleural nodules, for instance, new right lower lobe pleural nodule measures 2.3 x 2.2 cm (4/218) and enlarging left lower lobe pulmonary nodule measures 1.5 x 1.4 cm (4/209), previously 0.7 x 0.6 cm.
Bilateral pleural effusions with enhancing thickened pleura with interval removal of pleural drain. Persistent but decreased interlobular septal thickening and scattered atelectasis. Pulmonary opacities and edema have also improved.

Liver and biliary tree: Heterogenous enhancement, likely related to congestive hepatopathy, which limits evaluation for underlying masses, however there appears to be a new discrete mass or conglomerate of masses in likely segment IVb/5 (5/162) measuring 3.9 x 2.6 cm. Gallbladder is contracted. Biliary ductal dilation.

Spleen: No significant abnormality.

Pancreas: No significant abnormality.

Adrenal glands: No significant abnormality.

Kidneys and ureters: No significant abnormality.

Gastrointestinal tract: Small hiatal hernia. No dilated bowel.

Peritoneal cavity: No free fluid or air.

Bladder: No significant abnormality.

Pelvic organs: No significant abnormality.

Lymph nodes: Overall similar size and distribution of supraclavicular, axillary, mediastinal, hilar, and retrocrural lymphadenopathy. For example:
* Right paratracheal lymph node measures 2.2 cm in short axis (3/32), previously 2.3 cm
* Left supraclavicular lymph node (3/16) measures 1.4 cm in short axis, previously 1.3 cm in short axis
* Right hilar lymph node measures 2.1 cm in short axis, previously 2.1 cm

Bones and soft tissues: No acute finding or suspicious lesion. Postsurgical changes in the anterior abdominal wall. Improved anasarca.

Procedure Note

Kristen Nicole Bird - 10/07/2022

Formatting of this note might be different from the original.

CT CHEST ABDOMEN AND PELVIS WITH CONTRAST: 10/7/2022 10:15

CLINICAL HISTORY: 24 years of age, Male, pericardial epithelioid mesothelioma, status post pericardiectomy and Pleurx placement on 2/17/2022, here for follow-up -he is status post 2 cycles of chemotherapy and 3 cycles of immunotherapy with nivolumab and ipilimumab.

COMPARISON: 8/8/2022

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Lung parenchyma, pleura, and airways: Enlarging pleural nodules, for instance, new right lower lobe pleural nodule measures 2.3 x 2.2 cm (4/218) and enlarging left lower lobe pulmonary nodule measures 1.5 x 1.4 cm (4/209), previously 0.7 x 0.6 cm. Bilateral pleural effusions with enhancing thickened pleura with interval removal of pleural drain.
Persistent but decreased interlobular septal thickening and scattered atelectasis. Pulmonary opacities and edema have also improved.

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Pancreas: No significant abnormality.

Adrenal glands: No significant abnormality.

Kidneys and ureters: No significant abnormality.

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Peritoneal cavity: No free fluid or air.

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Lymph nodes: Overall similar size and distribution of supraclavicular, axillary, mediastinal, hilar, and retrocrural lymphadenopathy. For example:
* Right paratracheal lymph node measures 2.2 cm in short axis (3/32), previously 2.3 cm
* Left supraclavicular lymph node (3/16) measures 1.4 cm in short axis, previously 1.3 cm in short axis
* Right hilar lymph node measures 2.1 cm in short axis, previously 2.1 cm

Bones and soft tissues: No acute finding or suspicious lesion. Postsurgical changes in the anterior abdominal wall. Improved anasarca.

IMPRESSION:

1. New and enlarging pleural and pericardial nodules.
2. Heterogenous enhancement of the liver, likely representing congestive hepatopathy, which limits evaluation. However, there appears to be a new right hepatic mass measuring up to 3.9 cm. Consider liver MRI for further evaluation if clinically warranted.
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6. Improved interlobar septal thickening, edema, and opacities.

Michael Nguyentat MD discussed these results with Dr. Roy MD by phone on the following date and time: 10/7/2022 14:05

I have personally reviewed the images for this examination and agree
with the report transcribed above.

Signed"Final report"

Authorizing Provider	Result Type
Mohana Roy	IMG CT ORDERABLES

Marc E. Wolin, Esq.
mwolin@saiber.com
John M. August, Esq.
jaugust@saiber.com
SAIBER LLC
18 Columbia Turnpike, Suite 200
Florham Park, NJ 07932
Tel: (973) 622-8401

-and-

Joseph D. Satterley, Esq.
jsatterley@kazanlaw.com
Denyse F. Clancy, Esq.
dclancy@kazanlaw.com
KAZAN, McCLAIN, SATTERLEY & GREENWOOD
A Professional Law Corporation
Jack London Market
55 Harrison Street, Suite 400
Oakland, CA 94607
Tel: (510) 302-1000

Counsel for Claimant Anthony Hernandez Valadez

IN THE UNITED STATES BANKRUPTCY COURT
FOR THE DISTRICT OF NEW JERSEY

In re:	:	Chapter 11
LTL MANAGEMENT LLC,	:	Case No. 21-30589
Debtor.	:	

DECLARATION OF ANTHONY HERNANDEZ VALADEZ

Under 28 U.S.C. § 1746, I, Anthony Hernandez Valadez, declare under penalty of perjury as follows:

1. I am an adult over the age of 18 years and have personal knowledge of the facts expressed in this declaration. If asked, I could and would testify to the truth of such facts.

2. I submit this declaration to apprise this Court of what has transpired since it issued its Order Granting Limited Relief from the Automatic Stay.

3. Over four days in mid-September 2022, I testified about my life, use of Johnson's Baby Powder talc, and medical condition. As to Johnson's Baby Powder, I testified that I used the product daily, sometimes several times, as part of my hygiene routine. I also testified that applying baby powder created visible dust that I breathed.

4. Throughout my four days of deposition, I constantly experienced body pain, headaches, stress, and anxiety. Also, I used supplemental oxygen because of my breathing difficulties.

5. To date, I have undergone five cycles of immunotherapy. At one point, I went temporarily blind. Fortunately, my doctor prescribed me eye drops, and my vision returned to normal. However, this incident exacerbated my anxiety and stress.

6. My mental state has not changed. I still experience significant anxiety and depression. Talking about my current state triggers my emotions to the point where it seems I am having a panic attack. I am still in disbelief and shock that I have terminal mesothelioma.

7. Physically, this disease and any treatments related to it, including immunotherapy, still cause me to experience nausea/vomiting, poor appetite, chest pain and tightness, breathing difficulties, discomfort, fatigue, and body pain.

Under 28 U.S.C. § 1746, I declare under penalty of perjury that the foregoing is true and correct to the best of my knowledge and belief. I executed this Declaration in Merced, California, on December 14, 2022.

DocuSigned by:
3A27F890E614477...
ANTHONY HERNANDEZ VALADEZ

Exhibit 17

Kazan, McClain, Satterley & Greenwood
A Professional Law Corporation
KAZANLAW.COM

Joseph D. Satterley
jsatterley@kazanlaw.com

September 7, 2022

The Honorable Michael B. Kaplan
United States Bankruptcy Court, District of New Jersey
Clarkson S. Fisher Courthouse
402 East State Street, Courtroom #8
Trenton, NJ 08608

Re: **LTL Management LLC, Case No. 21-30589 (MBK)**

Dear Chief Judge Kaplan:

I write to provide you with a status update on the talc mesothelioma case of my client Anthony Hernandez Valadez and to respond to the letter from Debtor's attorney Dan Prieto (the "Prieto Letter") [Dkt. No. 2973]. Pursuant to this Court's Order Granting Limited Relief from the Automatic Stay, filed on August 8, 2022 (the "Lift-Stay Order") [Dkt. No. 2386 at p. 2], Mr. Valadez was granted an expedited trial date of November 7, 2022. To ensure that he and Protected Parties[1] are ready for trial on November 7th, if the Third Circuit reverses this Court's orders,[2] I request that this Court enter the attached Proposed Order. I agree with Debtor's counsel that we can address the Proposed Order at the omnibus hearing on September 14, 2022.

I. Mr. Valadez's California State Court Case is Set for Trial on November 7, 2022.

The California State Court has set Mr. Valadez's case for an expedited trial on November 7, 2022, if "the New Jersey Bankruptcy Court stay is lifted."[3] It also ordered the parties to "meet and confer to discuss stipulations for the extension of statutory deadlines with regard to discovery, motions, expert designations, authorizations, etc." and to seek this Court's assistance "as to what, if any, additional order is needed [for] the parties to adequately prepare this matter for trial by November 7, 2022."[4] These issues will be discussed in state court on September 22, 2022.

II. Despite the Lift-Stay Order, Protected Parties Have Refused to Prepare for Trial.

Now that he has a trial date, Mr. Valadez is preparing his case for trial in case the Third Circuit reverses this Court's orders. Thus far, Mr. Valadez has noticed his deposition, and

[1] As defined in the Lift-Stay Order.
[2] The following orders of this Court are on appeal to the Third Circuit: (i) Order Denying Motion to Dismiss [Dkt. Nos. 1572 and 1603]; and (ii) Order Granting the Preliminary Injunction [Dkt. Nos. 1573 and 1635].
[3] A true and correct copy of the State Court's Order Granting Trial Preference is attached hereto.
[4] *Id.* at p. 2.

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(510) 302-1000 • Fax (510) 835-4913

EXHIBIT 17

Protected Parties will attend it. Also, Mr. Valadez will work with Protected Parties on a stipulation regarding testing Mr. Valadez's pathology materials upon receipt from his treating facilities.

Beyond that, Protected Parties have rejected Mr. Valadez's efforts to ensure that all parties are ready for trial and that Mr. Valadez could appear and have his case tried before he passes. For example, I informed Protected Parties that Mr. Valadez intends to propound discovery on what efforts they have taken to preserve documents relevant to this case and depose any of their employees with knowledge about talc issues that plan on leaving or retiring before trial.[5] I also proposed reasonable discovery and pre-trial deadlines, including that all discovery must be completed one week before trial. I also met and conferred with Mr. Prieto and proposed that the parties agree to discovery. In response to each request, Protected Parties contend that such discovery is outside the scope of the Lift-Stay Order. Mr. Prieto also asserts that "[n]one of these requests, however, are necessary because [Mr. Valadez's] counsel already knows that the relevant evidence is preserved."[6] In order words, Protected Parties argue that, notwithstanding the Lift-Stay Order and the California State Court's order, they need not do anything to prepare for trial.

Protected Parties' position is unsurprising because they have consistently refused to resolve this matter or stipulate to lift the stay since they were given notice of this case on April 20, 2022. Indeed, Debtor is essentially asking this Court to reconsider the Lift-Stay Order when its counsel repeatedly asserts that requiring Mr. Valadez and Protected Parties to prepare for trial would be unfair "while thousands of similarly situated claimants remain subject to the automatic stay and the PI Order."[7] A hearing on this disguised motion for reconsideration is not appropriate.

III. Entry of the Proposed Order Ensures That the Parties Are Ready for Trial.

Because the parties are at an impasse and it is undisputed that there is substantial medical doubt about Mr. Valadez's survival beyond late November or early December 2022, Mr. Valadez requests that this Court enter the attached Proposed Order to allow the parties to take and complete the discovery needed to be prepared for trial. This Court has the power to do so because it retains jurisdiction to hear and determine all matters regarding the Lift-Stay Order.[8]

Mr. Valadez's request is also consistent with the Lift-Stay Order and this Court's oral order on July 28, 2022. In part, this Court found it "reasonable" and "makes sense for equitable considerations" to grant Mr. Valadez "limited relief to allow discovery" to "gather and preserve evidence that will support the claims against J&J in the future."[9] This Court hoped that the Lift-

[5] Footnote two of the Prieto Letter is irrelevant because the discovery Mr. Valadez proposed narrowly seeks facts specific to this case. [Prieto Letter at p. 2, fn. 2.]
[6] *Id.* at p. 2.
[7] *Id.*
[8] Dkt. No. 2836 at p. 3.
[9] 7/28/22 Transcript at 20:13-21:3.

Stay Order would "not…slow down the progress of the case Mr. Satterley presented."[10] Accordingly, entry of the Proposed Order is appropriate.

IV. Absent the Relief Requested, Your Honor's Oral Order on July 28th is Meaningless.

If the Third Circuit reverses either of this Court's orders, Mr. Valadez anticipates that Protected Parties will contend in state court that they need many weeks to be prepared for trial in this complex case because this Court's orders precluded them from conducting the necessary discovery. The Prieto Letter confirms this when it asserts that the Lift-Stay Order does not allow the parties to prepare for the expedited trial that Your Honor allowed them to obtain.[11] The entry of the Proposed Order cures Protected Parties' concerns because it will enable them to conduct the discovery required to prepare their defense. Beyond that, this argument lacks merit.

This Court should not allow Protected Parties to frustrate Your Honor's oral ruling on July 27, 2022, and to avoid preparing for the November 7th trial that Your Honor allowed Mr. Valadez to obtain. First, Protected Parties have been on notice of this case since April 20, 2022, and were given facts of this case, including medical records and declarations.

Second, Protected Parties have ample time to defend themselves at trial because of their years of experience litigating talc mesothelioma cases. Consistent with other such cases, Protected Parties will call an expert in mineralogy, likely Dr. Matthew Sanchez, to opine on the absence of asbestos in talc used in Johnson's Baby Powder. They will also call an epidemiologist, Dr. Richard Attanoos, to testify that epidemiological studies of talc miners and millers do not show an increased risk of mesothelioma despite the prevalence of mesothelioma in end users of talc. Protected Parties will likely have an expert, such as Dr. Gregory Diette, to opine that there are non-talc-related reasons for Mr. Valadez's mesothelioma. Protected Parties have advanced each of these defenses in almost every mesothelioma case.

Third, the state court is familiar with asbestos talc cases about Johnson's Baby Powder. and has already ruled on numerous motions in limine and page-and-line designations of key witnesses that the parties typically file in such cases.

Mr. Valadez respectfully requests Your Honor enter the Proposed Order to allow him and Protected Parties to take the necessary discovery to be prepared for trial on November 7, 2022. Absent the requested relief, the Lift-Stay order would become meaningless.

Respectfully submitted,
/s/ Joseph D. Satterley
Joseph D. Satterley

Attachments

[10] *Id.*

[11] Prieto Letter at p. 3; *see also* 7/28/22 Transcript at 20:13-21:3.

SUPERIOR COURT OF CALIFORNIA, COUNTY OF ALAMEDA

Rene C. Davidson Courthouse, Department 18

JUDICIAL OFFICER: HONORABLE JO-LYNNE LEE

Courtroom Clerk: Timothy Lopez CSR: None

22CV012759 August 26, 2022 10:00 AM

VALADEZ
vs
JOHNSON & JOHNSON, et al.

MINUTES

NATURE OF PROCEEDINGS: Hearing on Motion for Trial Preference

Counsel on Zoom:

Joe Satterley, Julia Romano, Ian Rivamonte, Noushan Noureddini.

REPORTED BY CSR SHEILA PHAM.

The Motion for Trial Preference filed by ANTHONY HERNANDEZ VALADEZ on 08/04/2022 is Granted.

Plaintiff Anthony Valadez's ("Plaintiff" or "Mr. Valadez") Motion for Trial Setting Preference pursuant to CCP §§ 36(d)-(f) is GRANTED pursuant to CCP § 36(d).

Plaintiff filed his Complaint in this action on 6/15/2022.

Plaintiff is only 23 years old but has malignant pericardial (lining of the heart) mesothelioma, a particularly rare form of mesothelioma. He alleges his claims against Johnson & Johnson and retailers for exposure to J & J baby powder (talc), which his mother apparently used regularly on him and in the home during his childhood.

Asbestos torts claims against Johnson & Johnson (or a successor entity holding Johnson & Johnson's asbestos torts liabilities) and retailers who sold Johnson & Johnson talcum powder products are currently stayed by the New Jersey Bankruptcy Court in the case In re LTL Management LLC, Case No. 21-3032. The New Jersey Bankruptcy Court stay Order is currently on appeal in the Third Circuit Court of Appeals ("Third Circuit").

Plaintiff successfully sought leave from the bankruptcy court to allow Plaintiff "limited relief to allow discovery, to gather and preserve evidence" and also "to make any appropriate motion required to get expedited trial dates ....in the event the Third Circuit does reverse ... and the case is dismissed."

Case 21-30529-MBK Doc 2079-5 Filed 09/07/23 Entered 09/07/23 15:08:36 Desc Main
Exhibit Exhibits 15-19 to Satterley Declaration Page 6 of 7

SUPERIOR COURT OF CALIFORNIA, COUNTY OF ALAMEDA

In support of the trial preference motion, Plaintiff presents the following evidence: Plaintiff's symptoms of persistent cough and pericardial effusion presented in 2020. He was given multiple courses of colchine and prednesone without positive response. On 1/4/2022, he presented with progressive cervical adenopathy (enlarged or swollen lymph nodes), worsening pericardial effusion and small pleural effusions. He underwent a biopsy on 1/10/2022, performed by declaring doctor Leah Backhus, his thoracic surgeon, pathology of which revealed pericardial mesothelioma, epitheliod type. In February, Mr. Valadez underwent bilateral thoracentesis with removal of 1.5 liters from each side of his chest.

Dr. Backhus declares that as of 2/14/2022, Mr. Valadez's "extensive adenopathy ... underscores the advanced stage of his pericardial mesothelioma." Another medical record of this date prepared by Dr. Backhus describes Mr. Valadez's mesothelioma as "overall poor prognosis with advanced local disease." On 2/17/2022, Dr. Backhus performed a partial pericardiectomy on Mr. Valadez and had catheters inserted in his chest. A medical record dated 3/8/2022 describes "diffuse tumor involvement of the pericardium with areas of invasion into the myocardium [heart muscle]." After the surgery, medical records indicate that Mr. Valadez still had "significant mesothelial tumor involvement around his heart and some into the myocardium."

Mr. Valadez's oncologist, Dr. Mohana Roy, provides a declaration reporting that Mr. Valadez began chemotherapy on or around 3/18/2022. According to Dr. Roy, Plaintiff underwent two cycles of chemotherapy as of 4/9/2022, which he did not tolerate well, suffering from mouth sores, linear dermatitis, nausea, lack of appetite, shortness of breath, acute anemia and fatigue. As a result, chemotherapy was discontinued after two cycles in May 2022. A 5/5/2022 CT scan showed that the chemotherapy was not effective: the CT scan showed "increased diffuse nodular interlobal septal thickening" and "new and increasing pulmonary nodules, concerning for progression of disease." As of 5/6/2022, it was decided that Plaintiff would instead undergo immunotherapy.

Dr. Backhus opines that there is substantial doubt that Mr. Valadez will survive more than six months from 5/22/2022, and Dr. Roy opines that there is substantial medical doubt that Mr. Valadez will survive more than six months from 6/2/2022.

Plaintiff's counsel declares that all defendants have been served with process.

Wherefore, the Court GRANTS Plaintiffs' Motion for Trial Setting Preference pursuant to CCP § 36(d)-(f). The jury trial shall commence on Monday, November 7, 2022 at 8:30 a.m. in Dept. 18, but only in the event that the New Jersey Bankruptcy Court stay is lifted as to defendant Johnson & Johnson and the retailer defendants prior to the 11/7/2022 trial date. The Pretrial Conference shall be held on the first day of trial.

Counsel shall meet and confer to discuss stipulations for the extension of statutory deadlines with regard to discovery, motions, expert designations, authorizations, etc. Where necessary, counsel shall obtain further clarification or further leave from the bankruptcy court as to what, if any, additional order is needed in order for parties to adequately prepare this matter for trial by November 7, 2022. These issues will be discussed at the next scheduled Case Management Conference.

SUPERIOR COURT OF CALIFORNIA, COUNTY OF ALAMEDA

The Court orders counsel to obtain a copy of this order from the eCourt portal.

Designated Defense Counsel shall serve all pending defendants in this action. Plaintiff shall serve endorsed filed copies of this order upon all defendants who have been served but not yet appeared or answered in this action.

By: T. Lopez, Deputy Clerk

Minutes of: 08/26/2022
Entered on: 08/26/2022

UNITED STATES BANKRUPTCY COURT
DISTRICT OF NEW JERSEY

Marc E. Wolin, Esq.
mwolin@saiber.com
John M. August, Esq.
jaugust@saiber.com
SAIBER LLC
18 Columbia Turnpike, Suite 200
Florham Park, NJ 07932
Tel: (973) 622-8401

-and-

Joseph D. Satterley, Esq.
jsatterley@kazanlaw.com
Denyse F. Clancy, Esq.
dclancy@kazanlaw.com
KAZAN, McCLAIN, SATTERLEY & GREENWOOD
A Professional Law Corporation
Jack London Market
55 Harrison Street, Suite 400
Oakland, California 94607
Tel: (510) 302-1000

Counsel for Movant Anthony Hernandez Valadez

In Re:

LTL MANAGEMENT LLC,

Debtor.

Chapter 11

Case No. 21-30589 (MBK)

SUPPLEMENTAL ORDER GRANTING LIMITED RELIEF FROM THE AUTOMATIC STAY

The relief on the following pages, numbered two (2) and (3), is hereby **ORDERED.**

THIS MATTER having been opened to the Court by Anthony Hernandez Valadez ("Movant"), by and through his counsel, the law firm of Kazan, McClain, Satterley & Greenwood, A Professional Law Corporation, and local counsel Saiber LLC, for the entry of an order supplementing the Order Granting Limited Relief from the Automatic Stay filed on August 8, 2022 ("Lift-Stay Order") [Dkt. 2836] as to non-debtors Johnson & Johnson, Johnson & Johnson Consumer Inc., Albertsons Companies, Inc., Lucky Stores, Inc., Safeway Inc., Save Mart Supermarkets, Target Corporation, and Walmart Inc. (collectively, the "Protected Parties"); the Court having considered the papers submitted on behalf of Movant and the Protected Parties; having heard the statements of counsel at the hearing held on September 14, 2022; and for good cause shown;

IT IS HEREBY ORDERED, ADJUDGED, AND DECREED that the Lift-Stay Order is supplemented as follows:

1. Movant and the Protected Parties be and hereby are granted further relief from the automatic stay and the Preliminary Injunction to conduct the discovery necessary for the parties to be prepared for trial in the Superior Court of California, County of Alameda ("State Court"), on November 7, 2022, in the event the Third Circuit Court of Appeals reverses this Court's orders enjoining and prohibiting Movant from prosecuting his claims against Debtor and the Protected Parties.

2. Pursuant to the State Court's request for deadlines on discovery and other related matters, this Court orders the following:

   a. All fact and expert discovery must be completed by October 31, 2022;

b. Parties are deemed to have demanded an exchange of expert witnesses and discoverable writings [Cal. Code Civ. Proc. § 2034.210, et seq.];

c. Parties must designate their expert witnesses and those witnesses' discoverable writings [Cal. Code Civ. Proc. § 2034.270] by no later than October 24, 2022;

d. The designating party shall "offer up" the expert(s) for deposition within a reasonable time before discovery closes;

e. Any party unilaterally canceling an accepted deposition will be responsible for re-calendaring the deposition within a reasonable time before discovery closes;

f. The parties are to meet and confer as necessary about a protocol for handling pathology evidence, including any destructive testing; and

g. All parties must respond within 14 days to written discovery (unless otherwise agreed to).

3. The stay set forth in Rule 4001(a)(3) of the Federal Rules of Bankruptcy Procedure shall not apply to this Order, and the Order shall be effective immediately.

4. The Court shall retain jurisdiction to hear and determine all matters arising from or related to this Order's implementation, interpretation, and/or enforcement, including with respect to the scope of the relief granted by this Order.

Exhibit 18

Order Filed on August 8, 2022
by Clerk
U.S. Bankruptcy Court
District of New Jersey

UNITED STATES BANKRUPTCY COURT
DISTRICT OF NEW JERSEY

Marc E. Wolin, Esq.
mwolin@saiber.com
John M. August, Esq.
jaugust@saiber.com
SAIBER LLC
18 Columbia Turnpike, Suite 200
Florham Park, NJ 07932
Tel: (973) 622-8401

-and-

Steven Kazan, Esq.
skazan@kazanlaw.com
Joseph D. Satterley, Esq.
jsatterley@kazanlaw.com
Denyse F. Clancy, Esq.
dclancy@kazanlaw.com
KAZAN, McCLAIN, SATTERLEY & GREENWOOD
A Professional Law Corporation
Jack London Market
55 Harrison Street, Suite 400
Oakland, California 94607
Tel: (510) 302-1000

Counsel for Movant Anthony Hernandez Valadez

In Re:

LTL MANAGEMENT LLC,

Debtor.

Chapter 11

Case No. 21-30589 (MBK)

ORDER GRANTING LIMITED RELIEF FROM THE AUTOMATIC STAY

The relief set forth on the following pages, numbered two (2) and (3), is hereby **ORDERED.**

DATED: August 8, 2022

Honorable Michael B. Kaplan
United States Bankruptcy Judge

EXHIBIT 18

THIS MATTER having been opened to the Court by Anthony Hernandez Valadez, by and through his counsel the law firm of Kazan, McClain, Satterley & Greenwood, A Professional Law Corporation ("Kazan Law"), and local counsel Saiber LLC, for the entry of an order granting Movant relief from the *Order (I) Declaring That Automatic Stay Applies to Certain Actions Against Non-Debtors and (II) Preliminarily Enjoining Certain Actions* [Dkt. 1635; Adv. Pro. No. 21-3032, Dkt. 187] (the "PI Order") as to non-debtors Johnson & Johnson ("J&J"), Johnson & Johnson Consumer Inc. ("New JJCI"), and retailers Albertsons Companies, Inc., Lucky Stores, Inc., Safeway Inc., Save Mart Supermarkets, Target Corporation, and Walmart Inc. (collectively, "Retailers" and with New JJCI and J&J, the "Protected Parties") pursuant to Section 362(d)(1) of the Bankruptcy Code, and waiving the fourteen day stay under Federal Rule of Bankruptcy Procedure 4001(a)(3) (the "Motion") [Dkt. 2348], and the Court having considered the Motion, the opposition filed on behalf of LTL Management LLC (the "Debtor") [Dkt. 2429], the reply filed on behalf of Mr. Valadez [Dkt. 2469], and the joinder in support of the Motion [Dkt. 2484], and having heard the statements of counsel and evidence adduced with respect to the Motion at hearings held on June 14, 2022 and July 26, 2022 (together, the "Hearings"); the Court having ruled at the June 14, 2022 hearing that Mr. Valadez was permitted to file a complaint to initiate an action, as defined in the Motion (the "California Action"), against the Protected Parties, and that, upon the filing of any such complaint, no action was required by any defendant named in such complaint pending a further ruling from the Court; and for the reasons set forth on the record at the Hearings and a hearing held on July 28, 2022;

IT IS HEREBY ORDERED, ADJUDGED AND DECREED that:

1. The Motion is granted in part and denied in part.

2. Movant Anthony Hernandez Valadez, having been authorized by this Court to commence a personal-injury lawsuit in the Superior Court of California, County of Alameda, be and hereby is granted relief from the automatic stay and the PI Order to permit Mr. Valadez to conduct such discovery as necessary to preserve evidence pertinent to Mr. Valadez's claim that may otherwise be lost or destroyed, including, but not limited to Mr. Valadez's pathology materials. Movant Anthony Hernandez Valadez be and hereby is also granted relief from the automatic stay and the PI Order to make any appropriate motion immediately in the California Action required to obtain an expedited trial date or priority in the event that the Third Circuit Court of Appeals reverses this Court's order denying the motion to dismiss the bankruptcy case [Dkt. 1603] and the case is dismissed.

3. The stay set forth in Rule 4001(a)(3) of the Federal Rules of Bankruptcy Procedure shall not apply to this Order and the Order shall be effective immediately.

4. Except as expressly provided herein, all provisions of the PI Order shall remain in effect.

5. The Debtor is authorized to indemnify and pay the Protected Parties for their costs associated with discovery authorized by this Order.

6. The Court shall retain jurisdiction to hear and determine all matters arising from or related to the implementation, interpretation and/or enforcement of this Order, including with respect to the scope of the relief granted by this Order.

Exhibit 19

SUPERIOR COURT OF CALIFORNIA, COUNTY OF ALAMEDA

Rene C. Davidson Courthouse, Department 18

JUDICIAL OFFICER: HONORABLE JO-LYNNE LEE

Courtroom Clerk: Timothy Lopez CSR: None

22CV012759 August 26, 2022
10:00 AM

VALADEZ
vs
JOHNSON & JOHNSON, et al.

MINUTES

NATURE OF PROCEEDINGS: Hearing on Motion for Trial Preference

Counsel on Zoom:

Joe Satterley, Julia Romano, Ian Rivamonte, Noushan Noureddini.

REPORTED BY CSR SHEILA PHAM.

The Motion for Trial Preference filed by ANTHONY HERNANDEZ VALADEZ on 08/04/2022 is Granted.

Plaintiff Anthony Valadez's ("Plaintiff" or "Mr. Valadez") Motion for Trial Setting Preference pursuant to CCP §§ 36(d)-(f) is GRANTED pursuant to CCP § 36(d).

Plaintiff filed his Complaint in this action on 6/15/2022.

Plaintiff is only 23 years old but has malignant pericardial (lining of the heart) mesothelioma, a particularly rare form of mesothelioma. He alleges his claims against Johnson & Johnson and retailers for exposure to J & J baby powder (talc), which his mother apparently used regularly on him and in the home during his childhood.

Asbestos torts claims against Johnson & Johnson (or a successor entity holding Johnson & Johnson's asbestos torts liabilities) and retailers who sold Johnson & Johnson talcum powder products are currently stayed by the New Jersey Bankruptcy Court in the case In re LTL Management LLC, Case No. 21-3032. The New Jersey Bankruptcy Court stay Order is currently on appeal in the Third Circuit Court of Appeals ("Third Circuit").

Plaintiff successfully sought leave from the bankruptcy court to allow Plaintiff "limited relief to allow discovery, to gather and preserve evidence" and also "to make any appropriate motion required to get expedited trial dates ....in the event the Third Circuit does reverse ... and the case is dismissed."

SUPERIOR COURT OF CALIFORNIA, COUNTY OF ALAMEDA

In support of the trial preference motion, Plaintiff presents the following evidence: Plaintiff's symptoms of persistent cough and pericardial effusion presented in 2020. He was given multiple courses of colchine and prednesone without positive response. On 1/4/2022, he presented with progressive cervical adenopathy (enlarged or swollen lymph nodes), worsening pericardial effusion and small pleural effusions. He underwent a biopsy on 1/10/2022, performed by declaring doctor Leah Backhus, his thoracic surgeon, pathology of which revealed pericardial mesothelioma, epitheliod type. In February, Mr. Valadez underwent bilateral thoracentesis with removal of 1.5 liters from each side of his chest.

Dr. Backhus declares that as of 2/14/2022, Mr. Valadez's "extensive adenopathy ... underscores the advanced stage of his pericardial mesothelioma." Another medical record of this date prepared by Dr. Backhus describes Mr. Valadez's mesothelioma as "overall poor prognosis with advanced local disease." On 2/17/2022, Dr. Backhus performed a partial pericardiectomy on Mr. Valadez and had catheters inserted in his chest. A medical record dated 3/8/2022 describes "diffuse tumor involvement of the pericardium with areas of invasion into the myocardium [heart muscle]." After the surgery, medical records indicate that Mr. Valadez still had "significant mesothelial tumor involvement around his heart and some into the myocardium."

Mr. Valadez's oncologist, Dr. Mohana Roy, provides a declaration reporting that Mr. Valadez began chemotherapy on or around 3/18/2022. According to Dr. Roy, Plaintiff underwent two cycles of chemotherapy as of 4/9/2022, which he did not tolerate well, suffering from mouth sores, linear dermatitis, nausea, lack of appetite, shortness of breath, acute anemia and fatigue. As a result, chemotherapy was discontinued after two cycles in May 2022. A 5/5/2022 CT scan showed that the chemotherapy was not effective: the CT scan showed "increased diffuse nodular interlobal septal thickening" and "new and increasing pulmonary nodules, concerning for progression of disease." As of 5/6/2022, it was decided that Plaintiff would instead undergo immunotherapy.

Dr. Backhus opines that there is substantial doubt that Mr. Valadez will survive more than six months from 5/22/2022, and Dr. Roy opines that there is substantial medical doubt that Mr. Valadez will survive more than six months from 6/2/2022.

Plaintiff's counsel declares that all defendants have been served with process.

Wherefore, the Court GRANTS Plaintiffs' Motion for Trial Setting Preference pursuant to CCP § 36(d)-(f). The jury trial shall commence on Monday, November 7, 2022 at 8:30 a.m. in Dept. 18, but only in the event that the New Jersey Bankruptcy Court stay is lifted as to defendant Johnson & Johnson and the retailer defendants prior to the 11/7/2022 trial date. The Pretrial Conference shall be held on the first day of trial.

Counsel shall meet and confer to discuss stipulations for the extension of statutory deadlines with regard to discovery, motions, expert designations, authorizations, etc. Where necessary, counsel shall obtain further clarification or further leave from the bankruptcy court as to what, if any, additional order is needed in order for parties to adequately prepare this matter for trial by November 7, 2022. These issues will be discussed at the next scheduled Case Management Conference.

SUPERIOR COURT OF CALIFORNIA, COUNTY OF ALAMEDA

The Court orders counsel to obtain a copy of this order from the eCourt portal.

Designated Defense Counsel shall serve all pending defendants in this action. Plaintiff shall serve endorsed filed copies of this order upon all defendants who have been served but not yet appeared or answered in this action.

By: T. Lopez, Deputy Clerk

Minutes of: 08/26/2022
Entered on: 08/26/2022